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ROYAL COMMISSION ON MATTERS OF HEALTH AND SAFETY ARISING FROM THE USE OF ASBESTOS IN ONTARIO

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180 Dundas Street,
Toronto, Ontario
Tuesday,
July 7, 1981
Volume XVI

ROYAL COMMISSION ON MATTERS OF HEALTH AND SAFETY ARISING FROM THE USE OF ASBESTOS IN ONTARIO

VOLUME XVI

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THE FURTHER PROCEEDINGS OF THIS INQUIRY
RESUMED PURSUANT TO ADJOURNMENT

APPEARANCES AS HERETOFORE NOTED

DR. DUPRE: Good morning, ladies and gentlemen. On your behalf and on behalf of the Commission, I am very pleased indeed to greet most warmly Dr. Henry Anderson, who comes to us from a sister jurisdiction in the great friendly nation to the south, the Wisconsin State Division of Health, some time with the Mount Sinai School of Medicine.

Dr. Anderson, your voluntary presence here is more than warmly greeted. We really appreciate your willingness to come in and offer expert testimony before us.

Counsel, are there any matters before I ask Miss Kahn to swear in the witness?

MR. LASKIN: Not for myself, but I could canvass my friends.

I think we are ready to go.

DR. DUPRE: Are we ready? Thank you.

Miss Kahn, could you swear in the witness, please?

DR. HENRY A. ANDERSON, SWORN

EXAMINATION-IN-CHIEF BY MR. LASKIN

Q. Dr. Anderson, we have a brief of your documents

MR. LASKIN: (cont'd.) which will be coming down shortly, and for the record we will file as exhibit twenty-three, so that if we have to refer to it or to any of your publications, we can simply refer to them by their tab numbers in the articles.

EXHIBIT # 23: The abovementioned document was then produced and marked.

MR. LASKIN: I gather you are going to give us the benefit of an opening address, with the use of slides?

THE WITNESS: Yes, I would.

MR. LASKIN: Good. Then I will retire to my usual place.

THE WITNESS: Could I have a pencil or a pen?

MR. LASKIN: Sure.

THE WITNESS: Thank you.

What I would like to do, for those of you who don't know who I am or what I've done, I have had, for a number of years, when I first came to Mount Sinai, my interest was in nonoccupational lung disease, or nonoccupational asbestos-associated disease, not solely restricted to dust diseases but to other occupational illnesses which can occur in circumstances other than occupational exposure.

As such, I would like to show you, as you will see in the documents that I have already published, some of the information that we have gathered on asbestosis or asbestos-associated diseases amongst nonoccupationally exposed groups, and then in my capacity as director of the Environmental Epidemiology Section for the State of Wisconsin, I am also in charge of our fledgling Asbestos in Schools project. Although we don't have a great deal of concrete data to share with you at this time, since we have only been in operation since October or November, we are beginning to generate some information and at least I can give you some firsthand experience as to how

THE WITNESS: (cont'd.) under one set of circumstances we have been able to inspect schools and at least give some of the results as to compliance in a situation where it has been voluntary at this point, as we do not have any mandatory regulations at this point.

If I may have the first slide, please?

This is primarily for focus, however I think it's important to keep in mind that there are a variety of illnesses, the first being asbestos...one which has led to some difficulty in diagnosis because there are a variety of factors that must be taken into account in making or establishing the diagnosis based on either clinical assessment, the chest radiograph, and of course tissue diagnosis being perhaps the most definitive.

Also, asbestosis was the first of the asbestos dust inhalation diseases. Subsequently, lung cancer, mesotheliomas and other tumors.

Next slide, please.

I think it's important to keep in mind, just for occupational health in general and most specifically the dust diseases such as asbestos-associated diseases, that we are dealing really with a variety of issues. One being the problem of past exposures, and unfortunately that...the results of those exposures we know the most about.

The difficulty is in assessing current exposures and trying to predict whether or not these represent safe levels or what the acceptable risk level is, and then of course we have concerns for future exposures or, in the case of asbestos, perhaps new uses that we, as of yet, have not found but which may appear.

Next slide.

I think also we have to learn from the past and one important thing to keep in mind is that there has been a great deal of optimism on behalf of the physicians, and if you go through the literature here starting with 1907, Montague Murray stating in his

THE WITNESS: (cont'd.) article where he described the first cases: "One hears, generally speaking, that

> considerable trouble is now taken to prevent the inhalation of dust, and so the disease is not so likely to occur as heretofore".

Of course we now know, some seventy-five years later, that this was somewhat wishful thinking, that although they had identified the source, or that inhalation of the dust was the causal factor in the disease and that there were means of controlling it, that getting it into the practical application so that when you are sitting in an office or room such as this it is fairly easy to say well, suppress the dust. But when you get out onto the mill or factory floor, that becomes a very difficult task at times, and we do need to keep in mind that the standard isn't what protects the health, but the actual level that's maintained at the exposure site.

So the lowest possible standard will do nothing to protect the health of the worker if in fact it is not enforced, or we are unable to attain it.

Next slide, please.

Just to go through the spectrum of the asbestos diseases, as my slide showed, the principal ones are asbestosis, which has a parenchymal or the fibrotic component, and the pleural component which is the scarring of the chest wall. There, of course, next are the neoplastic effects, the number one concern being lung cancer, which is the most prevalent, then the pleural and peritoneal mesotheliomas, and then a host of others which are of lesser significance in actual numbers of affected individuals.

There are other asbestos-associated diseases, benign pleural effusions, Kaplan's syndrome - combining asbestos dust disease with rheumatoid arthritis, and of course asbestos warts and then clubbing of the fingernails, which is nonspecific

THE WITNESS: (cont'd.) for asbestos, but in some cases is associated with the exposure.

Next slide, please.

Again, we have to keep in mind where we are today in perspective with where we were in 1907, and in the early years of the use of asbestos, and where you get up into Canada, and especially over in Quebec, of course, it becomes very important because there are a high proportion of miners and millers, but for the majority of the world, as you see on this slide, the asbestos producers can produce with very few number of individuals all of the asbestos which is used in the world, or coming out of Canada, of course, a large proportion of the chrysotile.

Then, of course, the processors...again, a relatively small number, nonlabour intensive type of factories generally.

Then we get to where the problem is today, or the principal problem, and that's in the asbestos product users, of which, if we look at a pyramid shape, the producers are at the top, the very small number, and as we go down the number of exposed individuals gets larger and larger, and now as disease has been described for some time in product users, that is where the principal concern lies and where the majority of the illness being seen has appeared. That's in the primary product users such as the insulators, brake repairmen, there are a number of others such as tapers in the painting trades who have used asbestos-containing materials no longer in use in the United States, and I don't believe, in Canada either...spackling compounds, joint compounds, that did contain asbestos.

The paraoccupational, we say, or bystander disease, this is the individual who may be working in the same work area, and as we all know, if we are in this room we all breathe the same air. If you open a bottle of perfume in one

THE WITNESS: (cont'd.) corner, it doesn't take long before you smell it in the other, and therefore the early concern of providing the actual asbestos worker with protective equipment, but the electrician or other construction people nearby, or utility and power plant workers who are not classified as being asbestos workers, they had a risk...I think you should all be familiar with the situation in the shipyards, especially during World War II, which, in the United States is probably now the principal source of the asbestos-associated diseases that we are seeing...railroad workers also must be included.

Then again, moving on, we have maintenance repair work. This is, when you go into any factory or most factories, often the layman is rather surprised to see that one-third to one-half of the employees are in fact maintenance. They have to keep these complicated machines and operations running. Whenever there is maintenance and repair, if the pipes have been covered or insulation has been used, it has to be removed to get at the joints or pumps, or whatever, and in refineries or anywhere where insulation, either for heat or cold, is needed, there is often asbestos found. Although replacement may not be with asbestos, we have a fine backlog of asbestos that's already in place, and of course then we take into account also public buildings which were sprayed, and the broad category of other industry where asbestos may be located.

My principal interest comes next, that of household contact. This is the dust, of course, can be brought home or escape from the plant in a variety of ways. It can be brought home by the worker, into the household, and as I'll show you, can be responsible for disease amongst family members.

We have public buildings, and then consumer products. As I'm sure you are aware in the last two to three years, the initial concern and the rapid removal from the market

THE WITNESS: (cont'd.) of the hair dryers and the asbestos in them, and of course asbestos in the schools now is also of concern.

So we have to be constantly aware of how materials are being used and although the construction engineer or the design engineer may find that an asbestos-containing material is the best possible for the use, often he is not skilled in assessing whether or not this will represent a public health hazard.

Next slide, please.

Just to very quickly show you, what I'm going to be talking about now is marker sorts of things that in public health or preventive medicine, we are interested in looking for either illnesses of conditions which will allow us to use them as markers. This is an indicator of exposure or an indicator of something significant going on.

One of those which has perhaps been described the most is pleural calcification. This is...here you can see on both sides of the chest extensive calcifications on the chest wall, and when it's bilateral, especially today in the absence of a history of other significant medical illness, almost exclusively is associated with some asbestos exposure.

Next slide.

There is also pleural thickening. Here you are helped out by an arrow. It should look like at the lower bottom there where you can see a nice, sharp rib edge, or up where the arrow is, you can see that there is a thickened area there showing what this pleura will look like in response to exposure.

Again, there are other causes, but the most common today is asbestos exposure, and almost invariably from the clinician's standpoint, the medical history will tell you whether or not this could be associated with a previous gunshot wound of the chest or a severe trauma. Generally this does not occur

THE WITNESS: (cont'd.) asymptomatically or old tuberculosis or other causes.

Next slide. please.

Here just shows a very severe, extensive case where you can see the thickened pleura all around the chest wall. Although generally a pleura is not associated with symptomotology or with restricted pulmonary function, in some cases it can become life threatening and verv severe, in which case an operation called decortication, where you strip off this pleura, can be of immense benefit to the patient. But this is a relatively rare occurrence.

Next slide.

Now, going back to our marker concept, looking at pleural calcification, this is a study done as a followup on insulation workers in 1976, of six hundred and fifty two individuals. I use this just to show the prevalence, or how common pleural calcification, pleural thickening, is in the occupationally-exposed group.

I apologize for not having put together slides of a more international list, but the same can be demonstrated from the mining and milling operations, as well as other types of factories. But here you can see that in the long-term workers, thirty-two, or one-third of them, had calcification, and over half had pleural thickening present, in this one study population.

So even if the numbers vary somewhat, you can see it's quite common and should be recognized by clinicians if they are educated sufficiently. I'll get into this a little later, but I think one thing that I have found is sorely lacking, and this probably is not the place to say it because everybody here is in the process of being educated, or is already knowledgeable in the field, but at least in the United States the general practitioner has heard very little about these diseases and is not terribly skilled or adept at making a diagnosis or even

THE WITNESS: (cont'd.) taking an occupational history, and I think in any kind of system for prevention if it is to be effective it must contain a very strong educational component. As we have more and more of these individuals, especially the shipyard workers, coming to their private physician, getting chest x-rays for perhaps a totally unrelated thing...be it chest pain for a heart attack or something like this...the physician needs to be attuned or educated to look for these sorts of abnormalities which may otherwise be overlooked.

MR. LASKIN: Can we just stop there? Is that the same plant from which you looked at household...

THE WITNESS: No.

MR. LASKIN: This is a different plant?

THE WITNESS: No, these are insulators.

MR. LASKIN: Okay. And...

THE WITNESS: This is a pilot project to a nationwide survey that I believe is just getting underway now, to see with insulators, because they are spread out, we hope to try to get them into central locations and this was a voluntary compliance sort of program.

MR. LASKIN: I see. So they don't come from any one particular plant?

THE WITNESS: No, no. These come actually from the southeastern part of the country, the U.S. Many of these are power plant insulators from the Tennessee Valley Authority area.

MR. LASKIN: Are they dealing with any particular kind of asbestos?

THE WITNESS: Well, whatever is in the insulation materials they are using. Generally they don't know the specific type of material they are working with, but it would be an end product user. I will later show you the plant that my household came from, which was virtually exclusively amosite asbestos.

THE WITNESS: (cont'd.) Next slide, please.

DR. DUPRE: Just for the record, I think that the slide that you are just showing is in the paper you coauthored with Dr. Selikoff in 1978?

THE WITNESS: Right.

DR. DUPRE: In our language, fortunately, you don't have to share.

That will be tab four, I believe, page 2498. The numbers check out.

MR. LASKIN: Thank you, Mr. Chairman.

THE WITNESS: I'm glad the numbers check out.

This is just another summary, hopefully a little easier to read than a large table. As scientists we are always accused of either putting up too much or too little information, and this one perhaps is too little, but it's again just to show the difference between Dr. Selikoff's 1965 study of insulation workers, the one he also conducted...or he and I conducted in 1978...and then again a separate group of talc workers from 1943, showing the calcification and parenchymal fibrosis in the populations. Here we have broken it out by duration from onset of exposure, less than and greater than thirty years.

I'm not sure that in the less than thirty year one I got the percentage right there, on calcification, but I think that should be in that same paper.

MR. LASKIN: It appears to be tab number eight, which is again the paper you did with Dr. Selikoff, entitled Asbestos-Associated Radiographic Changes. I believe it's table number fourteen dash three at page 263.

THE WITNESS: Next slide.

If you want to label all these, we are going to be here all day and tomorrow.

Now, with that as a backdrop just to show how common amongst asbestos-exposed, and in the specific instances

THE WITNESS: (cont'd.) of the tables, insulators, here shows a summary of the literature of the general population and the prevalence of pleural calcification.

I would also mention that we now have...which was not used in these various studies...have an international classification that even today, interpretation of x-rays is very much an art as well as a science. There has been a valiant attempt to objectify interpretation by the International Labour Organization out of Geneva, with the ILO classification, which has a series of standard chest x-rays with which the reader is to compare, and therefore we should be able to, in the future, compare studies which have used this classification.

In the earlier days of many of these studies when they took place, these were not read according to any standardized technique, and in fact many of them did not record parenchymal changes, and because when you are reading thousands of x-rays like these individuals...you can imagine a hundred and fifteen thousand x-rays...the vast majority of which are normal...that this can get to be a boring task and when something unusual pops up like a calcification, even though it was not part of the protocol, it was reported, although other changes were not.

So therefore we do have fairly good information going back, as you can see, to 1939, documenting the prevalence of pleural calcification in a variety of populations. You can see it jumps around depending on the area.

Can we go to the next slide?

MR. LASKIN: Can we just stop there for a minute so I can make sure I've got all these terms straight?

You've talked about parenchymal fibrosis, which I take it is the irregular opacities?

THE WITNESS: That's right.

MR. LASKIN: Then you've talked about a number

MR. LASKIN: (cont'd.) of pleural changes, pleural thickening?

THE WITNESS: mmm-hmm.

MR. LASKIN: Pleural calcification.

THE WITNESS: That's correct.

MR. LASKIN: The term asbestosis, as we have come to hear it in these hearings, does it encompass all of those things or any one of those things?

THE WITNESS: Well, generally...and again, as I say, the diagnosis of clinical disease is somewhat different than perhaps the epidemiologic diagnosis. That on the chest x-ray for the various studies that are performed, generally the pleural abnormalities, the calcification and thickening are considered to be asbestos-related, and in fact in the classification are quoted as such, as are the irregular opacities.

Depending on the clinician or the investigator, a pleural disease alone may be called asbestosis. Some may prefer to call it pleural asbestosis. Parenchymal disease is probably the...if we go back in time...the original asbestosis was parenchymal fibrosis, although in many cases pleural was described along with it. But generally asbestosis from the pathologic standpoint is fibrosis of the lungs. It may or may not have associated with it pleural disease, but in a broader sense generally the pleural disease is also considered asbestosis.

MR. LASKIN: When you say that, considered by whom? Because suppose this has diagnostic implications, it may have compensation implications in a sense, but we might have to make some determination of what is or is not asbestosis.

THE WITNESS: Well, if I can stick close to home I would say at Mount Sinai, where I came from and personally myself, I consider pleural changes to be asbestosis. From a compensation standpoint or from a disability standpoint, that's a different story. You then need to do a more thorough evaluation...the

THE WITNESS: (cont'd.) pulmonary function, the actual capabilities of the individual, go into at least my understanding of compensation. As far as whether or not a person is disabled, I think frequently this is a problem with the workers that if they are told, even if they have both pleural and parenchymal changes, that they have asbestosis, they think that they are immediately disabled.

This is not so. The vast majority of individuals, like those I showed on that last slide, with abnormal chest x-rays, can function adequately. So therefore there is a difference. You have to differentiate between what we are going to call disability, or from my standpoint anyway, compensable injury.

Now if you define injury as being evidence on a chest x-ray without attendant disability, then I think you could call pleural changes clearly are a demonstrable effect of asbestos inhalation. You have scarring, it's an abnormal condition, should not be there, therefore it is damage done by the inhalation of asbestos.

Parenchymal damage also can be done, and frequently they will be together.

MR. LASKIN: Dr. Uffen?

DR. UFFEN: Just so I understand. Since you are going to be speaking a lot about calcification, is calcification just calcium carbonate that appears on the lungs?

THE WITNESS: It's on the chest wall.

DR. UFFEN: But it is calcium carbonate?

THE WITNESS: I believe so, yes.

DR. UFFEN: Does it come from the process that makes bones, or does it come from irritant, whatever it is? Where does it come from?

THE WITNESS: We really don't know. It gets deposited, and there is calcification in this plaque which is

THE WITNESS: (cont'd.) collagen.

Now, just exactly how it comes about, it's probably partially an irritant effect.

DR. UFFEN: Is it identified and given a name by what you see in the x-ray, or is it given a name because it has calcium in it?

THE WITNESS: Well, it's both. You can generally see the calcium...and when I'm talking here, this is only, when I say pleural calcification, that's the calcification that can be seen on the chest x-ray. Frequently if you go to an autopsy, calcium or calcification can be found in a plaque, but not sufficient quantity or concentration to be seen on the chest x-ray.

So probably more plaques or pleural thickening may have some calcification in it than we can see on the x-ray.

DR. UFFEN: This may be naive, but does anything else cause calcification?

THE WITNESS: You can, if you have trauma. For instance if you have a hemothorax you can have the...and it isn't withdrawn right away...the blood clot can form and this can become calcified. If you also have a tuberculous empyema, empyemas can also have calcification in the pleura.

So generally it's a foreign body or something like that. As I say, those things can usually be ruled out.

Now, if you have somebody with multiple fractured ribs on one side and you see some pleural thickening, then of course...which is due to what...you would have to get an x-ray before the ribs were broken.

So there are diagnostic problems, but in the majority of cases, at least in the U.S., this is not the case.

MR. LASKIN: What is pleural plaque, which is another term that we've heard from time to time?

THE WITNESS: Okay. These are all terms that have

THE WITNESS: (cont'd.) come about and they are really a mixture of pathologic descriptive terms and then these pathologic tissue or histologic descriptions are translated into radiographic appearances.

Technically speaking, on the chest x-ray you are seeing shadows, and then we interpret them, are they fibroses in the lung...of course it is a shadow and it's speculation as to what it's due to, and by association we can generally make that case.

But the radiologists will like you, and that's why the terms are descriptive, irregular opacities, not fibrosis of the lung. Fibrosis of the lung is a tissue pathologic description where irregular opacities are a shadow on an x-ray.

Pleural thickening is what we see on the chest x-ray and can be measured, and it's just that - pleura.

Pleural plaque is usually used to represent pleural thickening, which is circumscribed. It's a small area.. if I had known I would have brought all my pathologic slides along and I could have shown you what these looked like.

So you can have diffuse pleural thickening, which would be that lung section I showed you. That was, of course, an exceptional case, but it can go all the way around.

The other x-ray that I showed that just showed kind of a little lump there, that would be a circumscribed plaque in the current nomenclature.

Then these plaques, probably in their natural progression, will go on, in many cases, to develop calcium or calcium deposits appear, and of course the calcium appears on the x-ray as a density, a bone density, and that's how we determine that there is calcification there.

So you compare it to what the ribs look like and say, I think there is calcification there. But it doesn't really...you can't predict a hundred percent of the time that

THE WITNESS: (cont'd.) it will be, until of course you get a pathologic specimen.

I don't know if that helps, but...

MR. LASKIN: Just one final question before we leave the point. You indicated that the Mount Sinai group considers that these pleural changes, other than whatever is meant by asbestosis, for generally all purposes I take it. Is it fair to say that that is somewhat of an issue as between various schools of medicine?

THE WITNESS: I think it has been. I haven't talked with enough people or read enough...or seen any altercations recently. I think it has been resolved by calling it pleural asbestosis as opposed to just asbestosis.

MR. LASKIN: Which would be the irregular opacities?

THE WITNESS: Yes. I think that most of the difficulties that...I would say yes, it probably can be an issue for some, and I wouldn't want to speak for the profession as a whole. We all read x-rays differently anway and make our own clinical diagnosis based upon many things. I would again separate between the epidemiologic interpretation of x-rays where you then have to assess false positives and false negatives, as opposed to a clinical evaluation. There's many other things go into...when I say a person has asbestosis, they must, of course, have had a history of asbestos exposure. You would like to see that.

So there are various grades of it.

Okay, next slide.

This again just shows several other studies with fairly high prevalences of pleural calcification. These were published, again, representing populations with environmental and neighborhood asbestos exposure, and in 1960 was one of the early papers. Dr. Kiviluoto, from Finland, as part of a TB,

THE WITNESS: (cont'd.) tuberculosis, survey was reading x-rays in two different areas and he found that the pleural calcification came virtually in toto from one area of Finland where there was anthophyllite asbestos in the soil, as well as a mine and mill in that area.

The reports from Bulgaria and Turkey also relate to asbestos or asbestiform minerals, I should say, in the soil or rock outcroppings in these areas, and that was the author's explanation for the high prevalence of pleural calcification.

MR. LASKIN: For the record, that slide is table fourteen dash two, at tab eight, page 263.

THE WITNESS: Next slide, please.

This just is a further breakdown of that, showing the total numbers examined and the definitions of the group, and you can see some additional work from Czechoslovakia, where in fact three groups were studied...the factory workers themselves, individuals in neighborhoods, as well as family members, and this was one of the studies which sparked our curiosity in the group that we studied.

Next slide.

This just shows the breakdown of those populations in Czechoslovakia, ranging from the general population rate of roughly point three percent pleural calcification, up to the neighborhood seemed to have the highest, but not really different from the factory workers or the relatives, all of which were at least a tenfold increase over the other areas.

Again, this is just a...using pleural calcification as a marker to indicate exposure. What this means or how this can be translated into disability or subsequent disabling or fatal disease is not well understood. But it does indicate or is a mechanism to identify individuals who are likely to have been exposed to asbestos, and therefore may have an attendant risk.

Next slide.

MR. LASKIN: Again for the record, that slide was table fourteen dash four in the same article, at page 264.

THE WITNESS: Now, this one I'm going to now speak briefly to mention another marker, which you have probably heard about, and that is the pleural and peritoneal mesotheliomas, and this is a summary of a fairly recent paper by Cochrane and Webster from South Africa, where they reviewed a series of seventy mesotheliomas that were admitted to their hospital. They interviewed them and got their histories when they were still alive. As I'm sure you are aware, people with this disease generally do not live very long and frequently in our mortality epidemiologic studies trying to go back and determine whether or not exposure may have taken place, after the individual is deceased and may have been deceased for many year, ten, twenty, thirty years, then trying to locate next of kin or somebody who may remember whether he was exposed, that as we get away from the individual themself the reliability of the information we get goes down substantially.

Here you can see that in this one series virtually all, or sixty-nine out of the seventy, had demonstrable asbestos exposure. This, of course, is the outside, nearly one hundred percent series. Many others, as you will see going through the literature, have less success at identifying asbestos. But it does show that certainly the majority of individuals with this disease have had an asbestos exposure in the past.

Next slide.

Now again one of the early studies looking at mesotheliomas was done by Dr. Newhouse and Thompson, from the London Hospital, and here you can see they reviewed all of their hospital files from 1910 onward, and identified a series of controls. You can see here roughly half, or thirty-one, had industrial asbestos exposure. Nine were relatives, eleven lived within a half a mile of the factory, and in twenty-five they were

THE WITNESS: (cont'd.) unable to identify an asbestos exposure.

Now, from my standpoint I was most interested in the figure there of nine relatives, and the neighborhood potential exposure, and that this may well have indicated a significant exposure in the homes as well as in the industrial site.

Next slide.

Now, I apologize. This is old, this is...I'm not sure which...I think it's my very first paper in 1976, when I reviewed the world literature that I could get my hands on, and I found there were some thirty-seven mesothelioma cases attributed to household asbestos exposure. That number has since probably close to doubled, and I haven't kept up with my slide production. But this just shows or goes to demonstrate that reports have come out of many different countries from around the world. You can see many are individual cases, and of course many have gone unreported.

Next slide.

MR. LASKIN: Again, that was table one, tab two, page 312.

THE WITNESS: Perhaps of interest on this slide is that series of thirty-seven cases broken down by type, being pleural, the majority - twenty-four of the thirty-seven. Four were peritoneal, and nine the report did not specify. You can see there is a clear predominance...and I believe that has been maintained...for pleural mesotheliomas in nonoccupationally-exposed groups, and you can see the predominance of females. This may well be artifactual because in most cases, by definition, the male of the household would have been the exposed worker and there were far more male asbestos workers than there were female, so that the reverse, the husband getting the disease while the wife was the worker, is less apt to have occurred.

MR. LASKIN: Is there some biological or medical

MR. LASKIN: (cont'd.) explanation for the fact that the environmental or nonoccupational mesotheliomas more often appear to be pleural?

THE WITNESS: I would say there is speculation, but I don't think we have any...at least I'm not aware of any definite explanation as to why that is.

MR. LASKIN: What's the speculation?

THE WITNESS: Well, it could be that there are relatively small amounts, or the exposure is relatively low, and that the fibers are in the lung and then they penetrate through to the pleura and the first line of defence, the macrophages, etc., in the lung, may well clear a lot of asbestos out and migrate to the pleural space, and getting the asbestos there. Where if you have a very, very heavy exposure, or even not so heavy but continuous exposure, a lot of the dust is cleared up out of the lungs and then swallowed, and would either get into the intestinal tract or you have an opportunity for more fibers and some of them to migrate and go through the diaphragm into the abdomen.

But as I say, what makes sense does not necessarily...what seems logical, perhaps, is not necessarily what in fact occurs.

It may well be that it's much easier to make a diagnosis of a pleural mesothelioma than it is peritoneal, and therefore just artifactually, since most of these are from death certificates or other cases, if somebody has a rapidly developing diffuse abdominal tumor, frequently they will not be operated on because by the time they come into their physician he says, this is far advanced, I can't do anything, and the peritoneals are not diagnosed. Where the pleural disease, frequently they will become symptomatic and they will see it and attempt some therapy.

So a lot of this can just be an artifact.

THE WITNESS: (cont'd.) Unfortuately, we don't have one hundred percent autopsies on everybody who dies, so we don't have many of these diagnoses.

Next slide, please.

This is a crude approximation just to use mesothelioma as a marker, and just for simplicity's sake we took the occupational asbestos work and put it at a factor of ten. As you've probably had testimony, depending on the population studied the percent of individuals dying from one to ten or thirteen percent in the occupational group.

But these are just rough approximations and on the righthand side is the potential for control. You will notice we have tried to avoid the optimistic predictions that others have made in the past and been proven wrong, but this is why we state potential there. At this point we have most information on mesotheliomas in the occupational, primarily the secondary exposed group, which as I say is the very largest. The vast majority of individuals exposed to asbestos today are those outside of the mining and milling operation.

DR. UFFEN: Could I ask you a question?

THE WITNESS: Sure.

DR. UFFEN: Is a mesothelioma caused by the fibers that remain in the lung, or could it be caused by fibers that have passed through the lung?

THE WITNESS: I don't think we have that answer. We do know in animals, you put it in and the fibers are there and then the mesothelioma occurs. But we don't know whether or not if the fibers are all dissolved in a very...you know, exposures many, many years ago, whether you have...whether the physical presence is necessary in order to have the disease.

In general you cannot find asbestos bodies or asbestos fibers in the tumor. Probably because it's a very fast growing thing and the asbestos bodies are in the lung around

THE WITNESS: (cont'd.) the tumor site.

MR. LASKIN: Can you explain what that slide is? What is the exposure category intending to show?

THE WITNESS: The exposure category is...this is coming from Dr. Nicholson's work, again on a rough basis...just to give a proportion as to the various types of measurements on a scale. As I'm sure you've heard, it's very difficult to compare fiber counts in an occupational setting to nanograms per cubic meter in the outdoors or general population.

But this is what they worked out from the meager measurements that have been made as to rough orders of magnitude. The idea is that in the general community the amount of fibers present are well...you know, multiple factors below what... especially in the past when the mesotheliomas being seen today occurred.

MR. LASKIN: Are the first four numbers then in fibers per cubic centimeter? And then the last two in nanograms?

THE WITNESS: No, it's supposedly in rough approximation.

In other words, you shouldn't try to interpret this into a measurement. It is a relative exposure index, so that we are not saying that these are the levels found, but they are in...you know, you can multiply them by anything you want... but this is the rough comparison between the two. That it's very hazardous to try to compare one type of measurement to the other and obviously exposures are going to vary by the area, and this is just as a comparison.

DR. UFFEN: Could you recall a reference where this relative scale would be defined or set out? So that we could look it up later?

THE WITNESS: I would have to check. This comes from Dr. Nicholson. This is one of his slides, and I would think

THE WITNESS: (cont'd.) it probably is a summary. I'm not exactly sure which one of his papers.

DR. UFFEN: I don't recall it having emerged in two days' previous discussion with him.

THE WITNESS: Not in this exact measurement.

DR. UFFEN: We spent a little bit of time on that very subject, so I'm not to interrupt any longer now, but...

THE WITNESS: Yes. I think if we go to the next slide I'll show you what it came from. I hope there is a reference here.

Yes. See text, so I'm not sure. It's one of his papers. I could look it up.

But this is more difficult to interpret than it is to put a number down a lefthand column like this, but this just shows the various ranges.

I think this is from the 1978 New York Academy paper, and this is in nanograms here. But you can see it ranges rather extensively, depending on the samples made. So it's impossible to pick one number, although you can pick a midrange figure. But that's why I don't, on that other slide, like to assign it anything because the next step to that is interpreting it in terms of a standard, and I don't think the information that it came from warrants that strict interpretation. It's more to give a rough idea in using what's available on counts to look at mesotheliomas, and we know the rates in the various communities, of mesothelioma...and the rates of mesothelioma do seem to fall out by the estimated exposures.

Next slide.

Now, this is the mortality experience of the workers in the amosite factory whose household members I studied, or we studied at Mount Sinai. One of the reasons for selecting this particular population was because...this should be in one of those papers too...there was an unusual mortality experience.

THE WITNESS: (cont'd.) We did know that the workers themselves had asbestosis, had radiographic abnormalities, and it was a relatively unique type of exposure in that it was solely amosite asbestosis and it was sufficiently in the past... 1941 to 1945...for us to perhaps anticipate seeing some abnormalities in family members, if in fact they were going to occur.

This just gives the observed to expected ratios for lung cancer, GI cancer.

MR. LASKIN: You've taken it to 1974 in that slide...

THE WITNESS: Yes.

MR. LASKIN: ...and I take it...

THE WITNESS: There may be a newer one.

MR. LASKIN: ...the corresponding table to 1977 is table fourteen dash five, at tab eight, page 264.

THE WITNESS: Yes. So the numbers you will see in the new table there, comparable, just larger numbers.

MR. LASKIN: Am I correct that this...I think Dr. Nicholson gave us some evidence about this plant. This is the plant at Paterson, New Jersey?

THE WITNESS: That's correct.

MR. LASKIN: As I recall, this was the plant where a number of the employees had very, very short work histories?

THE WITNESS: Yes.

MR. LASKIN: But very intense exposures for short periods of time?

THE WITNESS: Yes. The next slide I'll show you that.

This was, you just mentioned, a wartime industry. It was, by description anyway, a very dusty, dirty factory, and individuals, since it was a defence industry, it was a draft-

THE WITNESS: (cont'd.) deferred industry, and individuals would often go to work here for very short periods of time either for a deferrment or because there was a fair amount of turnover, while they were waiting to be inducted they would work there.

As you can see, they started between 1941 to 1945. Some did in fact work the full...the plant closed in 1954, so thirteen years was the longest period, a relatively small number of individuals worked for that whole period. But you can see there is a fair proportion of those who worked for one, two or the majority of individuals less than one full year in that plant.

Because the numbers are relatively small, this is for the death of lung cancer and again I believe there is an updated table of this in the recent Annals, in Dr. Selikoff's work. The numbers have increased but the ratios and the basic observations remain the same.

This comes from Dr. Seidman's work, too, again in the New York Academy Annals.

But you can see that even in the very short-duration individuals that there is an increased observed to expected ratio for lung cancer in this group.

Again this is a group that was followed through 1974, so this is, in many cases, many years after their exposure.

Also unique to this plant is, the majority of these individuals were not asbestos workers or this type of factory worker by trade, and therefore when they left the employ of this factory they frequently went into some other business and did not have subsequent asbestos exposure. So in that sense the majority of these individuals were unique, and I would refer you to Dr. Seidman's paper on that.

It offered a relatively unique experience, to be able to assess short exposures with no subsequent exposure, and

THE WITNESS: (cont'd.) the long-term outcome of that.

Next slide, please.

This now, to show you some of my work, and again slides have a way of appearing, or primarily disappearing, so you are going to see a combination here of two papers. The numbers do not change drastically, but the latest included, I believe, an additional examination which had fifteen more individuals in it, so the numbers total will vary between seven seventy-six and seven eighty-five, I believe.

But this just shows the relationship and the number examined up to the point of this slide. You can see the majority were sons and daughters, as you might expect.

Next slide.

This just shows when the individual is first exposed into the household. Four hundred and forty came from the 1941 to 1945 era, and the remaining from the 1946 to 1954, and you can see we have duration of employment. That's the index worker. You of course have no good measurement or assessment of exposure in the household from that period of time, and one again, to create an index for comparative purposes, we did look... we had two numbers available, the one of the lefthand side there, when they were first exposed or duration from onset of first exposure, and then the length of time that the worker worked in the plant. You can see nearly half of the family members were associated with a worker who worked less than one year in that plant.

What I don't have here and we don't have a firm handle on, is how long the family member may have remained in that house once the asbestos dust got into the house, with reentrainment when they would vacuum or dust. This may have had a significant impact on subsequent disease.

But this is what we would consider active contamination of that house.

DR. DUPRE: Dr. Anderson, could I ask a question that was on my mind as I read this progression of figures?

THE WITNESS: Sure.

DR. DUPRE: As I understand it, the cohort of household members that you were looking at starts at a size of three hundred and twenty-six when you are working on it back in your 1976 paper...

THE WITNESS: Right.

DR. DUPRE: ...and then it gets to six hundred and seventy-nine, I believe, in your 1979 New York Academy of Science paper. Then it gets to seven seventy-six, I guess in the most recent one?

THE WITNESS: Mmm-hmmm.

DR. DUPRE: Now, one point that just left me somewhat confused was the following: You refer in your 1976 paper, at page 312, in this instance not to radiographic abnormality, but to deaths from mesothelioma.

THE WITNESS: Right.

DR. DUPRE: Now, these deaths, as I understand it, of course did not take place within your cohort that grows from three twenty-six to...

THE WITNESS: Not the clinical cohort, right.

DR. DUPRE: ..to seven seventy-seven?

THE WITNESS: Right.

DR. DUPRE: So this is found where? It is found in the relatives of the one thousand, six hundred and sixty-four workers?

THE WITNESS: That's correct.

DR. DUPRE: That were your original index workers?

THE WITNESS: That's right.

Perhaps if I give you a little history of the project in itself, it would help.

DR. DUPRE: That might help us.

THE WITNESS: What we have is, we have, as you mentioned there, the thousand-odd workers. Now...

DR. DUPRE: One thousand, six hundred and sixty-four who worked from 1941 to 1954, as you describe it.

THE WITNESS: Right. So we have the names of those workers. The first group, the workers employed 1941 to 1945, which is nine thirty-three, was the original group that Dr. Selikoff started tracing.

Now you have to remember that this plant closed in 1954, and it's some twenty years later that we are not only trying to find where the man was, and he may have worked one month and subsequently left. And we are not only trying from my standpoint to find the worker, but once we find the worker to find out who lived with him at the time he worked there.

So when you speak of a cohort, we have...and we are nearly completed with that now...a mortality study of a cohort which is going to include all of the family contacts that we could identify from these workers...which is going to be some three thousand-odd individuals.

Once we identified those people and determined that they were in fact in the household, that they were alive, they were then invited to come in for a clinical exam. Obviously we couldn't make them come in, so as you see the numbers increase, what this shows is, as we trace not only the workers, but identified the family members and also found the family members, we then invited them to come into a clinic.

Now the workers were being followed serially, too, which made it somewhat easier...at least for those workers who were alive. But obviously those who were deceased, we then needed to trace next of kin and try to find out. It's very difficult to do.

So as we went along we invited individuals as we identified new individuals, to come in. That's...we have nearly

THE WITNESS: (cont'd.) exhausted the list, at least to invite people to come in. So that's where the numbers change.

In other words, the mesothelioma deaths that occur are from the universe which is now nearly defined as to how many family members there were, from the whole group. We then have the clinical group, which is a different subcohort, and we don't of course know, other than looking at the worker, whether or not the group who came in is different from the other. What we do know is the workers, or the ones that came in, tended to be from the workers who were easier to find, which tended to be the longer-employed workers. We have fewer one-month workers, or those one-month workers may not have lived with anybody...a lot of rooming house people.

So our cohort, in quotes, that total and family population at the time of the first study was unknown. But as we identified them, they were invited in and they came in.

DR. DUPRE: Let me see if I can follow this. Could I just ask you to turn to page 312, in your tab number two?

THE WITNESS: Right.

DR. DUPRE: As I take it, what is being described here is that there were...if I look at the first full paragraph, it starts with the word 'among'...there were two mesothelioma deaths already identified?

THE WITNESS: Right.

DR. DUPRE: So they could not have formed part of the cohort?

THE WITNESS: Not of the clinical exam, no.

DR. DUPRE: Of the clinical cohort.

THE WITNESS: I mean that's among the group already traced.

DR. DUPRE: Right.

THE WITNESS: Right.

DR. DUPRE: Now, let's go to the bottom paragraph?

> "In addition, two living cohort members have biopsy-proven pleural mesotheliomas and are currently in therapy".

THE WITNESS: Right.

DR. DUPRE: Now is that a part of the clinical cohort?

THE WITNESS: Well, they were identified during the clinical study, yes. They are now, subsequently, dead.

DR. DUPRE: That, of course, I guess, is what is followed up at page 398 of tab seven, if we could just take a look at that.

Do you see the paragraph at the top that begins, "Using only"...etc.?

THE WITNESS: Yes.

DR. DUPRE: When you get to the bottom of that:

> "In our previous report we described four instances of pleural mesothelioma from among the total household contact cohort under observation".

THE WITNESS: Right.

DR. DUPRE: Is that four the two that were already identified as having died, on page 312?

THE WITNESS: That's correct.

DR. DUPRE: Plus the two to which we have just referred...

THE WITNESS: That's right.

DR. DUPRE: ...that were identified under the clinical program?

THE WITNESS: Right.

DR. DUPRE: Then when you say, back on page 398, "An additional case has been detected," now is that fifth case within your clinical cohort?

THE WITNESS: No. Well, it was identified during

THE WITNESS: (cont'd.) the clinical cohort, but it is an individual who had prior asbestos exposure. So technically... it is a family member, but it was a worker at the factory...

DR. DUPRE: Who had a direct exposure as opposed to domestic exposure?

THE WITNESS: No, had previous domestic exposure, so we felt that we could not attribute it to exposure coming out of this plant. This was a man who helped set up this factory, who had worked ten previous years in England, and that's where the child was born and raised. It happened also...the child, I should say...I think she was thirty years old when he was working in Paterson, she lived with them, came under the rubric of the definition of the cohort, but in getting the history you could not attribute it to this plant.

Everybody else here, to the best of our ability, did not have previous domestic exposure or occupational exposure.

So that we have five domestic cases, but technically speaking only four of them would be included as coming out of this cohort from the plant.

DR. DUPRE: And only two of them were actually identified as part of the clinical diagnosis?

THE WITNESS: That's right.

DR. DUPRE: Thank you.

MR. LASKIN: Can I ask you, just before I forget about it because you said you are doing a mortality study now, are you able to say at this stage whether these household contacts are showing any excess lung cancer, for example?

THE WITNESS: At this point, as I said, we are just finishing the tracing, and no, I don't believe we can. There obviously have been lung cancer deaths, but it does have to be standardized against New Jersey rates, and we have not got all that inhouse yet.

MR. LASKIN: Are you able to say whether your

MR. LASKIN: (cont'd.) mortality study is going to show any deaths from asbestosis?

THE WITNESS: I can't specifically state that, no.

Next.

Now, as you would expect, and what is very difficult from the mortality study to eliminate, are individuals with known occupational exposure. As in any general population you would expect some of them to have had occupational exposure. During the clinical evaluation they were in fact queries as to their lifetime exposure, or possible exposure, and individuals who may have had exposures either to asbestos or other pneumoconiotic dust were listed here and eliminated from the subsequent analysis because obviously any abnormalities seen could well be attributed to their trade rather than to their household exposure. Since our records in tracing generally gave the year and perhaps the month that the individual worked, some individuals were included in the study or came in for examinations who were either then found to have a birthdate which was a few weeks to months after the man stopped employment, or a relative was listed, a sister or brother was listed as living in the household, by the worker, but in fact had only come after the man had stopped work. There were some forty of those, and therefore technically they were not part of the cohort and they were also eliminated.

So that comes to a total of ninety-one. You can see at the bottom there the breakdown of that.

Next slide, please.

Just to show what the x-rays showed of those who had occupational exposure, you can see direct asbestos trades - there were some nineteen, and overall this group, twenty-three of the fifty-one were abnormal, for a forty-five percent rate.

This is anyone of the x-ray abnormalities, either irregular opacities - category one over zero or greater, pleural thickening - either side, pleural calcification, as well as

THE WITNESS: (cont'd.) diaphragmatic pleural plaques. We did not consider loss of the costa phrenic angles or obliteration of the angles as being specific enough to warrant inclusion. If we had done that, there would have been a potentially higher rate.

So those were the abnormalities in the ILO coding that are associated with asbestos, and have been included. Individuals such as this we then took out because the abnormalities could be attributed to something else and what we wanted is to try to only have individuals whose known exposure could have been in the household.

Next slide, please.

MR. LASKIN: That last slide is tab seven, table one, page 389.

THE WITNESS: So this is just a summary group slide. As a comparison, because you always need to have a comparison group, we chose urban New Jersey residents who happened to be attending the clinic where the others were being examined, and therefore we had chest x-rays taken on the same equipment over roughly the same period of time, which made a masking of x-rays somewhat easier, that the technique in all was the same so you could not readily as a reader whether a film was a control film or a study film.

Here you can see there were three hundred and twenty-six of these. We did review their clinical charts and since this was a clinic of which Dr. Selikoff was a member, in the majority of cases there was at least a brief occupational history for the individual in the control group, and we tried to eliminate from the control group any individuals who may have had occupational exposure. As I say, we did not interview these individuals, but did review their clinical charts.

You can see that group had roughly five percent abnormal films, as read by this classification. One could say

THE WITNESS: (cont'd.) that that represents the background level seen in a similar urban population. As you were saying, from a disability or a compensation standpoint whether those would represent some, quotation marks 'asbestosis' or not, since they have no known exposure, these could well just be false positives because there is a rather extensive variability in what is considered normal.

We have those who entered the household, those forty that in the subsequent tables that have been removed, but it is of interest that the group is still very small. These were individuals who lived in the house, many of them for a number of years after the man stopped working, and could well have been exposed to dust that remained there and was recycled into the air.

DR. UFFEN: The ILOUC pneumoconiosis classification, does that exclude other kinds of illnesses like we commonly call pleurisy, or something like that?

THE WITNESS: No. It's a descriptive code. Now you have opportunity when you are looking...see, you don't know anything about the film, so you do have a series of other codes that if you, for instance, think the abnormality you are seeing is due to something else, say it looks like carcoid, now it is irregular opacities but it has a pattern that just doesn't look like asbestos and you think, this guy has got something else, there is another code that allows you to do that.

There were, I think, some six or seven individuals, the numbers were so small, that had other medical explanations, from the six eighty-five there, that were removed. They happened to have normal x-rays, but by reviewing the medical histories we felt had to be eliminated because...

DR. UFFEN: Some other...

THE WITNESS: Yes, even though they were normal. After we knew they were normal, we couldn't decide to include them. We had to make that decision ahead of time.

DR. UFFEN: What is going through my mind is, years ago I remember we always had little skin tests. If you got a positive response, they popped you in front of an x-ray machine.

I always got a positive response and I would hate to get my lifetime cummulative dose of x-ray exposure, and I never ever have been able to find out why. All I knew, there was a spot on my lung, but they were...

THE WITNESS: Well, generally that would be... probably what you had was what we call Ghon complex, which is...

DR. UFFEN: Goon or ghon?

THE WITNESS: Ghon, G H O N, which represents an old primary focus of TB. In other words, at some point you were exposed, you were skin test positive, and there is a little calcified on your x-ray which is inactive.

DR. UFFEN: The same kind of calcification as...

THE WITNESS: No, no.

DR. UFFEN: No?

THE WITNESS: No, this is parenchymal. This is in the lung tissue itself, as opposed to on the pleura, or the wall. That we don't even record for this ILO system. That's so relatively common that that is not recorded and there is never any problem differentiating between those.

DR. UFFEN: Pardon the interruption, but we are getting so many terms that it's hard to relate to them.

THE WITNESS: Absolutely.

DR. DUPRE: Dr. Anderson, while you are being generous on the subject of interruptions, could I just once again go back to what our dialogue ascertained a few moments ago: namely that two individuals who were identified within the clinical cohort as having mesothelioma, and of course wound up dying from the disease. Now, I'm looking at page 395 on tab seven, and I'm looking at the bottom table, table twelve. This may be either an idiotic or an unfair question, or maybe both, but I'm

DR. DUPRE: (cont'd.) asking it, being both idiotic and unfair.

Would you have any recollection, because one of the mesothelioma cases was a daughter, if I remember rightly, and the other was a son, okay?

THE WITNESS: Mmm-hmm.

DR. DUPRE: No, one was a sister-in-law, the other was a son. But do you have any recollections, just to help me out, as to where these two mesothelioma victims would have been classified in terms of their radiographic abnormalities? Would it have been...

THE WITNESS: They wouldn't have been...they weren't included here.

DR. DUPRE: Oh, you would have taken them out?

THE WITNESS: Yes. They are coded as CA or cancer, and the one, by the time we saw these folks, they had...they would have been classified as being malignant. In that case, you don't read it as benign pleural thickening.

DR. DUPRE: Okay.

THE WITNESS: So had we not suspected a malignancy, they probably would have been in the pleural abnormality category.

So anytime there is, there were some that had been operated on, say for lung cancer, and had radiation treatment, those are the kind of people that you just can't list them all. They were just taken out.

DR. DUPRE: Okay. Just again to take table twelve as an example, they would not be included in the six seventy-nine that appears on the bottom at the lefthand side?

THE WITNESS: No, no.

DR. DUPRE: Okay.

THE WITNESS: At least they weren't supposed to be.

So here you can see we are left then with six eighty-five. This is an update on the six seventy-nine. I think

THE WITNESS: (cont'd.) also here you will see that there is...yes...

MR. LASKIN: It's table number seven, at tab eight, page 266.

THE WITNESS: Yes.

They didn't change much. We just had a handful more that we felt make the tables different.

So you can see here that if we assume that the New Jersey urban residents represent either background or a very low level residential exposure, the percentage of abnormalities on the x-rays increases with exposure. Again, with the very low number of individuals who are in that second category of forty, obviously although statistically they are different from the residents, you would be much more comfortable attributing that to a significant difference if we had more numbers.

So it's sort of an interesting observation at this point, which needs to be elaborated on and we are trying to get more of those type of people in.

Next slide, please.

This shows the ILO classification of both the six eighty-five, after all those others were removed, and the three twenty-six control group or comparison group...I shouldn't call them controls. Anything to the right of the double line is what we consider to be abnormal.

As you can see, there were very few of the family members that had parenchymal abnormalities or these irregular opacities in an advanced or a high-grade. The two over two would be considered by most individuals to be moderate parenchymal asbestosis, where the one over zero is usually considered a borderline. As you can see, in the control group zero one, one over zero group, accounts for the majority of individuals who were in the comparison group that had anything but an absolutely perfectly normal film.

MR. LASKIN: Is there any relationship between the degree of abnormality on the films and ordinary disablement?

THE WITNESS: Well, generally there is an association that the denser the fibrosis, the greater the likelihood that you will have a physical impairment. But you cannot...although that's a general statement, again you have to evaluate each individual. You can have a man that has an x-ray that looks like he has no lung left at all, and he's just fine. Then again you will have somebody with a moderate abnormality...that you would say this is a run-of-the-mill case...who has severe disease as measured by diffusion or pulmonary function. He is disabled.

So you can't...a lot of this will depend on how long he is from his exposure, how long the x-ray has had a chance to so-called develop. In other words, the clinical picture appears on the x-ray. You can have damaged lungs, but if it's early on, you may first see the pulmonary function abnormalities, because the x-ray changes or your ability to detect it on x-ray has not progressed sufficiently.

MR. LASKIN: I take it from your articles that basically these people when you examined them were essentially healthy?

THE WITNESS: That's right.

MR. LASKIN: Do you anticipate that over their life that because of their asbestos exposure that they are going to become sick or disabled? Are you able to say?

THE WITNESS: Well, one never likes to try to predict. Certainly you have concern. We do know that it is a progressive disease and that there will be change over time. Whether or not all of these individuals will go on to be disabled, I think you can say no. The likelihood for any one of them to reach some disability is probably low.

But are they at increased risk? I would have to say yes. I mean, I would certainly rather be an individual with

THE WITNESS: (cont'd.) a perfectly normal x-ray than to be forty year old individual with some early irregular opacities and pleural disease, because you just don't know who is going to be the one that may go rapidly, and who is the one not, and obviously the less your exposure, the less likelihood that it will progress rapidly.

MR. LASKIN: As between your report in 1976 and your report in 1979, did you re-examine any of the same people so as to make any assessment as to whether there has been progression of abnormality?

THE WITNESS: Well, we have re-examined some of them, especially some of the wives. I don't think we have a large enough number that I can say definitely, but some of them there have been changes, or when the x-rays were read, the new x-rays were read, they were graded what would be considered a higher grade.

But I don't think that we've seen a sufficient number of those with abnormal films to start with, to be able to say that this represents a significant difference. The pleural thickening may be a little thicker, but whether or not that has any significance, we don't know. The people still feel the same, but there have been some changes. But I would not call them, at this point anyway, significant.

Next slide.

MR. LASKIN: That last slide was table eight, the same article, at tab eight.

THE WITNESS: Again here you have the...you are getting a brief course in ILO U/C classification, and here is the pleural thickening. If you will look at this slide and sort of mentally remember the last one, you will see that the predominant abnormality that we saw was pleural. That in this group of individuals there was very little parenchymal or irregular opacities, there were very few irregular opacities

THE WITNESS: (cont'd.) present, and when they were present they were in the lower grades. This was not necessarily the case with the pleural thickening or the pleural calcification.

Here is what I was saying, potential progression. We really don't know because you measure it at one time width A, which is up to five millimeters, and they come back the next time and you put your ruler up and it's seven millimeters, which puts it into a B category. Are we going to call that progression or not?

So that's where we are at at this point, but you can see extent two means it runs pretty much greater than one-half of the projection of the lateral chest wall. There weren't too many of those, roughly three percent overall had that type or extensive abnormality.

Next slide.

Now this again is an attempt to put the figures to indicate that the pleural abnormalities were again the predominant observation. Here you can see that the only abnormality present for a hundred and twenty-six of these, or eighteen percent, was pleural.

In other words, their irregular opacity reading was zero one or less, which would be considered normal parenchyma.

The others, there were sixty-one, or nine percent, that had only the irregular opacities without pleural, and fifty-three, or roughly eight percent, had both pleural and parenchymal abnormalities and would represent perhaps the classic appearance, or more classic appearance of what we know as asbestosis.

Next slide, please.

MR. LASKIN: The last two slides are tables thirteen and nine, for the record, in the same article.

THE WITNESS: Now, although the observation, the overall observations are interesting, which leads...or what leads

THE WITNESS: (cont'd.) credence to those observations was our ability to be able to see some sort of a dose response, even though we had no measurements of the dust in the homes and our only estimates were the length that the man worked there, and therefore the period of time which he may actively have brought workclothes home or dust home in any number of ways.

I think you can appreciate that...you've probably had considerable discussion on how do we translate nanograms or weight measurements or particles to fibers. At least there you have a measurement. Here we just have a very crude approximation of exposure. Of course one man's practice, how clean he was, what he did, may differ and we could not take that into account, so this is a very crude approximation and we made up for the crudeness of that by having the larger numbers of individuals included.

So as you can see here, for the three hundred and fourteen who had contact with the worker for less than one year, overall twenty-six percent had an abnormality and you can see irregular and pleural abnormalities, it was about evenly spaced. Fifteen percent, if you now go to the relatively small number, our thirty-five individuals who had a worker there for more than ten years, you can see over half of them had abnormalities and again you can start to see how the pleural abnormalities pop out, with roughly twice as many as in the irregular opacities present.

I would note that the categories of one over one are greater for the irregular opacities, were predominantly seen in the longer duration group.

Next slide.

DR. DUPRE: Just a question on the lefthand column there.

THE WITNESS: Go back.

DR. DUPRE: What that is measuring is the duration

DR. DUPRE: (contd.) of the employment of the index worker?

THE WITNESS: That's right.

DR. DUPRE: So it is not necessarily measuring the number of years in which a household contact was exposed to that worker?

THE WITNESS: Yes. That's what it is. In other words, if a worker was there, there were very few instances where a child would have been born into a house of a ten-year worker after he had worked five years. If that were the case, that would have been, exposure would have been coded as five years.

So you can't always compare, you know, the years to the length of the work, but the duration of employment, just to keep the title low, was the duration of active employment of the worker that the person was exposed to.

DR. DUPRE: As opposed to the number of years that the person was exposed to?

THE WITNESS: As opposed to the number of years that the worker was there. In other words, the worker may have been there much longer, but the person in the top column...the worker could have worked there for ten years, but less than one year duration of employment, we are considering that the family members was exposed to that worker for less than one year of his work in the plant.

DR. DUPRE: I see. So let's say someone...your index worker was in the plant fifteen years?

THE WITNESS: Right.

DR. DUPRE: But had a child who left home two years after he started employment. Now, that child shows up as a figure in the one-to-five years?

THE WITNESS: Right. And the wife might well be in the ten-plus.

DR. DUPRE: Okay.

THE WITNESS: Which makes it...you can start to see the ramifications.

DR. DUPRE: Well, it's the duration of the ...

THE WITNESS: The family member's contact with the worker.

DR. DUPRE: The family member's contact.

THE WITNESS: Right.

DR. DUPRE: Rather than the duration of employment of the index worker.

THE WITNESS: Right.

MR. LASKIN: That's table eleven, for the record.

THE WITNESS: It's sometimes hard to get the words out.

Okay, next slide.

Now, here we have taken the other look and that's year of onset, and again the exams were done, would range roughly twenty-five to thirty years from their onset of exposure. You can see the very early group, forty-one to forty-six, and this is the group that we know the mortality of the workers, those that worked after that period it has been much more difficult to identify the workers because it was after the war and some of the wartime security measures which allowed us to identify the worker were no longer in practice, and therefore the records that we had to go on to find the worker so we could identify the family were just not there, and that's the group we are working on now.

But here again you can see the various prevalences of abnormality, and perhaps the most striking is the difference in the 1941 to 1946 group, in the pleural thickening and pleural calcification, compared to the group that was 1950 to 1955.

MR. LASKIN: Again for the record, that's table eleven of tab seven, page 395.

THE WITNESS: Next slide.

Okay, this...I think you are beginning to see the complexity of working the various exposures, and then we have the various relationships. As you can see here, this is broken down by wives, daughters, sons, siblings and others. What was interesting and we are trying to explain, and we don't have a good explanation at this point, as you can see the overall prevalence difference between the daughters and the sons...the sons having roughly twice the prevalence of abnormality of the daughters. Yet, as you see, their mean duration of exposure is approximately the same.

In fact, all of the various groups came out to have very close to the same mean duration of exposure. Overall the wives had the highest prevalence of abnormality, and you can see as it goes down there.

Next slide.

This is the x-ray status, normal, abnormal, by the various clinical findings. As you can see, there was a paucity of clinical abnormalities. Five individuals had clubbing, as you can see four out of the five had abnormal x-rays, the same with cyanosis. As you can see, those...the cyanosis also went with the clubbing, and so the same four individuals appear in all four columns there. So these are not mutually exclusive categories.

Then you can see that the abnormalities are...the clinical abnormalities are more prevalent in those with the abnormal x-rays than with the normal x-rays, but again you do have these clinical findings in those with normal chest x-rays, and these are not symptoms or physical findings that are unique to asbestos exposure alone.

MR. LASKIN: The rales in the first column, is this the same thing that the British have used as an index of asbestosis?

THE WITNESS: Right.

MR. LASKIN: That's the thirty-five figure out of six eighty-five?

THE WITNESS: Right, right.

And I would also, just for the epidemiologists, the examining physicians, although they could obviously see the age of the individual and they examined him, did not know what their x-ray looked like when they did the exam. So they were blinded to that.

Now, as I say, it was fairly obvious which were the...in most cases anyway, which were the daughters and which were the wives, and obviously you can tell a son from a father, so you are blinded only to the extent that the physical person in front of you tells you what he is by looking at him.

So, next slide.

One of the things I saw, and that you were interested in, and that was the potential effects or additive effect of cigarette smoking. What I've done here...this is not yet published...but I did break out the cigarette smoking categories by never having smoked...they reported never smoking cigarettes, that's two hundred and fifty-six; former cigarette smokers, a hundred and one; current cigarette smokers, three hundred and twenty-five. This is in that family group, and whether or not pleural abnormalities were present or the prevalence in the smoker category, as well as irregular opacities, and one or more abnormalities, and you can see that the pleura abnormality is virtually the same in all three groups, although the smokers tend to have, percentagewise, less than the former smokers. Irregular opacities were more prevalent in the current and former smokers, nearly twice as prevalent as in those who never smoked cigarettes.

What we haven't done yet is take into account duration of their exposure and other conditions like that, although crudely there did not seem to be great differences

THE WITNESS: (cont'd.) between the smoker groups.

MR. LASKIN: Is that what you would expect, no changes between smoking and nonsmoking groups, for pleural changes?

THE WITNESS: I don't know what you would expect. I guess what has been described is that there hasn't been an effect on pleural abnormalities, and what has generally been seen is an additive smoking effect. Again, the explanation, we don't know for sure, but what would seem to be the logical explanation is that perhaps the smoking in someway impedes the expiratory mechanism and therefore more fibers will remain in the lung. You are overloading with other particles and therefore you don't clear as well. We have no way of knowing that for sure.

There are some animal studies on clearance mechanisms that are being studied, to see about that.

As I say, this is an interesting observation but at this point I don't know that the differences here may well be explained by the duration of exposure or the relationship of the person. What we do know is that many of the older women did not smoke cigarettes, and they tended to be the wives and the mothers.

Although, as I say, we have not corrected for that here, but it would seem to...exposure would probably be weighted towards the nonsmoking group.

Next slide, please.

These are the four current cases, and I would also, just to explain a little bit more, as far as how we assigned a relationship, there were frequently more than one worker living in the household. In other words, a father and a son may have worked, and what we did in that case...and those are other analyses that are currently underway...is to see whether or not we should add those exposures. In other words, if a person lived in a household where two people worked at the same time, each

THE WITNESS: (cont'd.) for five years, should that represent ten years, should it represent five years? At this point we have taken the relation..we have used the relationship of the worker with the longest duration of exposure. So that in some of these, the wife may well also be a daughter-in-law, or something like this. I'm not sure in these cases here.

Next slide.

Now, this is another table that again has not been published because the total household contacts are still not well defined. At this point we had roughly two thousand contacts.

Again, this is the mortality cohort, of which some of them have been examined. So here you can see the comparison between the worker group, the nine thirty-three that has been studied, and they so far have developed fourteen mesotheliomas, versus the four in the household contact group.

MR. LASKIN: Are there enough numbers there to say one way or the other as to whether the amount of dose, I take it, the household dose would be less has any effect on the latency period for mesothelioma?

THE WITNESS: As you say, the numbers are very small, and with four individuals you have four points. It's hard to say that. It would seem, looking at this, that clearly the mesotheliomas that we've seen, of which there have only been four, occurred thirty years-plus from their onset, where for some of the workers by that point there had been a larger number.

Whether or not that represents a significant increase in latency period, it could well be that. If we had seen them earlier, you couldn't say that at all. But again, we are following this group and were one to appear in the twenty to twenty-four year group, obviously your pattern would be completely destroyed. So you can see with small numbers, just by adding or subtracting one...especially adding one anywhere along the line there...can destroy how you would interpret it.

THE WITNESS: (cont'd.) So you have to interpret it with a great deal of caution.

DR. DUPRE: Is that table in one of the articles?

THE WITNESS: No, no.

MR. HARDY: Mr. Chairman, perhaps we should take copies of the slides that have not been published, to add to the record, if that's possible.

MR. LASKIN: Can we arrange to do that, Dr. Anderson?

THE WITNESS: I think we probably can, sure.

MR. LASKIN: I'll arrange to circulate to the parties copies of the slides that aren't in the material.

DR. DUPRE: You don't have any data yet about the fifth case of mesothelioma to which you refer in the article?

THE WITNESS: Oh, yes. We have all the information. I just don't want to put it in here because it would be misleading.

I mean, it really doesn't belong. It would fit into the thirty-five-plus group there, but thirty-five years-plus from her initial exposure. If you took it from her exposure in this plant, it would go in the twenty-five to twenty-nine group, you see, because she had been exposed ten years before.

We can't say that the mesothelioma wasn't caused by her household contact while he worked at this plant, but conversely we can't say that it definitely was. So it remains a household case, but not another one.

Okay. Next slide, please.

I think now, some of these are slides that aren't mine and if you are going to want...I'm not sure whether some of these have been published. I think we probably better..I don't feel that I can make them copies for the record, so we'll probably skip right over them.

THE WITNESS: (cont'd.) They come from the Cancer Society, but I would rather have them release them than me, so why don't you skip ahead about three, four slides.

Ooop, nope, we can go back. These you should have, I believe. No, let's go through these and get up to the...keep going...keep going.

Okay, this has been published. This is in the New York Academy paper and may be of interest. This is from Dr. Selikoff's work on the insulation workers, again pointing up the problem of competing risks, and this is among the twenty-two hundred and seventy consecutive deaths. Obviously in that number there will be some individuals who have more than one tumor present at the time of their death, and therefore it may not be listed on the death certificate as the cause of death, but was found incidentally in the medical record or at autopsy.

Here you can see there were one hundred such neoplasms that were present, but there was another cause of death. You can see that this would increase the number of lung cancers, and in fact there were three mesotheliomas that were present but were not the immediate cause of death of the individual. Colon cancer is also fairly high up.

Next slide, please.

Now, this is just back on information. Since you were interested in the effect of cigarette smoking, and I don't know how much you have been presented again, this is not asbestos worker, but this is just to show from the American Cancer Society what is known about cigarette smoking and lung cancer mortality ratios, by the type of smoking and the increased risk.

Next slide.

Now, this shows, again from Dr. Selikoff's work in the seventeen thousand insulators, this is in the twelve thousand individuals who are twenty or more years from onset

THE WITNESS: (cont'd.) of employment, and compared to nonasbestos workers who never smoked, and that comparison group is taken from the American Cancer Society's smoking study that was begun in 1959.

Here you can see amongst the insulators of those with smoking history known, and then by whether they smoked twenty-plus or one pack a day or more...these are U.S. packs... and then those who smoke less than. You can see the observed-to-expected ratios are relatively high, with eighty-sevenfold observed-to-expected ratio for those in the twenty-plus, compared to thirty-six in the former smokers, and the never smokers are at fivefold increase.

Next slide.

This just summarizes that and is perhaps a little easier, since we are going rapidly here.

For the nonsmoking asbestos worker, there is about a fivefold increase in the lung cancer rate compared to other nonsmokers, but in real actual numbers terms, since the rate is so low in the nonsmokers that the actual numbers or increase, fivefold increase, still leaves a very small number of workers. But in fact they do have a fivefold increased risk.

Now, of course, in the cigarette smokers where the cigarette contribution is very high for lung cancer, when you multiply that by five it gives you a great number of excess deaths. This is, of course, a group where education is extremely necessary.

Next slide, please.

This again just compares the control Cancer Society group with their death rate per hundred thousand of roughly eleven point three for the nonsmoking, nonasbestos-exposed person. The asbestos worker who doesn't smoke cigarettes, you can see the rough fivefold ratio increase there - fifty-eight deaths, or forty-seven per hundred thousand. For the smoking,

THE WITNESS: (cont'd.) nonasbestos, you can see there is a tenfold increase, so that the cigarette component there can be very strongly seen, and then in the asbestos worker who also smokes, you can see the rate is up fifty-three times, or to six hundred per hundred thousand.

Next slide, please.

Again, this is a summary slide looking at the various types of tumors which have been associated with cigarette smoking, and those who have not, and here you can see that cigarette smoking, at least in this insulator group, did have an influence on the lung cancer rate; did not affect either of the mesotheliomas; did affect esophageal cancer; did not stomach, colon, kidney cancer; did for larynx and pharynx and buccal cavity. And there was an influence on death due to asbestosis.

Next slide.

Now, of course, what everybody wants to know is if I stop smoking, will it do me any good. This again is from the Cancer Society just showing what happens in the general population, nonasbestos workers, and again expressed as age standardized death rates, with current smokers at one thirty-seven. What's interesting is to see that those who have stopped less than a year nearly triple their death rate, so obviously you don't want to stop smoking.

However, I think also logic would dictate, and those of you who know somebody who has developed a cancer or had a heart attack, will know that it's at that point that they stop smoking and their longevity from their underlying illness is severe.

So in that first year, we have these competing risks and you can see from that point on it begins to drop off. At ten years, having stopped, their rate is roughly down at what the nonsmokers is.

Next slide.

THE WITNESS: (cont'd.) For stopping smoking...again this is looking at the insulators only...if you stop smoking the risk decreases to one-half to one-third of those who continue to smoke.

Next slide...this should be the last one, I think.

This is again from the New York Academy of Dr. Hamman's, showing that the standard observed-to-expected ratios begin to go down. Now, this is a standardized figure, but you can see the importance of stopping cigarette smoking as it goes down...those who are less than five years, those who are five to nine years, and those who are ten years plus...goes down significantly, but does not quite reach the never-smoked group, which has the standardized, normalized ratio of one.

Next slide. I think that should be it.

Oop, that reminds me. Okay, now I'll just briefly go over what we are doing. I'll switch gears and go to our Asbestos in Schools program, and of course the whole issue of environmental asbestos...you can turn the slides off now.

I don't know if we want a break or...

MR. LASKIN: Take a break? Sure.

DR. DUPRE: Shall we take a five minute break, until 12:20?

MR. LASKIN: Sure.

THE INQUIRY RECESSED

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THE INQUIRY RESUMED

MR. LASKIN: I think we are ready to resume, Mr. Chairman.

Dr. Anderson, you are going to deal with your involvement in the monitoring of asbestos in the schools in Wisconsin?

THE WITNESS: Yes. What I've done is I've brought

THE WITNESS: (cont'd.) a number of things along with me which I'll go over briefly.

In the United States, the EPA is approaching regulations governing testing or identification of asbestos in schools, and as part of their initial process, each of the States was asked to develop an Asbestos in Schools plan, and I'll leave with you what our plan is, but essentially what it is is to inspect all the various schools using a scheme developed by the EPA to identify the, whether or not that school had asbestos in it, and then, as you may have a copy here, a little exposure assessment profile which ranged from the primary factors being the condition of the material...this is done by an inspector. In Wisconsin, the Department of Industry, Labour and Human Relations has building inspectors that go out for other reasons and they were impressed into duty to inspect the various schools, or the schools themselves can hire or have trained appropriate individuals, and part of the assessment is the condition of the material, whether or not there has been water damage, how much exposed surface area there is. A great deal of importance is how accessible it is, the activity and movement in the area.

In other words, if this is in the basement where the only person who goes there is the maintenance man, this has a different treatment than if it's in a classroom or a corridor or in a gymnasium or a cafeteria where the majority of the children may go, and we also assess whether or not there is any evidence that there has been damage done.

I suspect Dr. Nicholson may have showed you his photos of what can be done to a ceiling by exuberant high school or younger children who want to see how high they jump and how far they can stick their finger into the ceiling.

Also of course what's important is how friable the material is, and what this little exposure factor score here does is to begin to try to assess how likely a problem is, and make

THE WITNESS: (cont'd.) recommendations as to how to alleviate that problem.

That can range from strictly noting that it's there, and labelling it, as would be done with the pipe insulation, and leaving it in place with periodic inspections to see that it is in good repair, to either encapsulation...which is taking an impervious compound and spraying it over the material so that it can't break lose and drift down...to removal completely.

Just in our inspections in Wisconsin, we have twenty-one hundred and thirty-four public schools, of which we are now inspecting at roughly the rate of fifty per month. There have been some fifteen hundred that have been done.

We have an additional nine hundred and some-odd nonpublic private schools to which, under our current statutes, we do not have right of access, so inspections have been somewhat slower from that standpoint.

Recommendations have been made and we have done return visits to approximately a hundred and twelve schools so far, primarily to schools where a recommendation for corrective action was made, and we have been very pleased that seventy-five of them have demonstrated compliance with the corrective action that was advised.

I would say, again, at this point there is no legal obligation on their part to follow the recommendations that are made and so at this point we are very pleased to see that action is being taken and that the efforts to go in and identify asbestos sources have in fact been heeded, and there does seem to be a co-ordinated plan on the part of most of these schools to begin to take care of potential problems.

Just one other quick thing. Part of the program is also an education program, and I'm sure probably in this area, as well as others, the loca PTA's or action groups become very concerned when the asbestos is identified, and push for immediate

THE WITNESS: (cont'd.) removal or action, often very costly and precipitous-type action. This also became of concern in Wisconsin, but the implementation of this program has gotten things back on a more even footing and we do feel that there is a more rational approach to rushing in. We have also had a very active education program, of which I'll leave the materials here, which explains to the school board, as well as others, just what is currently available, what is known about asbestos, what the relative risk may be as vis a vis occupational type exposures, which is what most of the general public has heard about, and therefore the relative exposures are considerably lower in the schools, and I think Dr. Nicholson's information that he presented probably showed you when you can identify using a scheme such as this, what appear to be from an inspection standpoint hazardous conditions, you can in fact, when you sample, find higher levels than in areas where you would not anticipate a problem. For that reason, we do not do any air measurements or anything like this in the schools. It's not part of our recommended procedure. We base it strictly on an inspection and a bulk analysis with optical staining and polarized light identification of asbestos in the bulk product that comes in.

As you can see that goes into the system, so it's a relatively crude sort of system, but we do feel you can identify at least areas that would be of higher risk from those where no action needs to be taken.

We have also found that there needs to be education of the contractors who have sprung up to try to pick up the load of what do we do about our problem here, and frequently they will have workers who are inexperienced with the removal or handling of asbestos products, and it's very important to see that the worker who is going to remove this, or alleviate a potential problem in a school, is also adequately protected.

This has not always been the case, and in some

THE WITNESS: (cont'd.) of the buildings removal, to save money, has been done by inexperienced or summertime students, and in order to cut costs you rip it out dry, try to do it on a weekend, and there really is no inexpensive way to safely remove the asbestos. There are some ways that are better than others, and of course the question becomes, is it safer to leave it in place than it is to inappropriately break it loose and perhaps spread it around the school in the process of removal.

So while we are hopefully alleviating one concern, we must not lose sight of the importance of maintaining the protection of the new group of employees that have to take it off. So we are also aiming our education program at this group to impress upon individuals who have not worked with asbestos that care and caution is needed, even if it's on a temporary basis.

We have a little slide/tape presentation that we go around to the general community to give them some basic information, and help explain to them why the complaints of colds, runny nose, etc., cannot be attributed to this agent and that there is no need to go in and do extensive evaluations of students in the schools, that the risk at this point, or the ability to detect the problem, just is nonexistent at this point.

MR. LASKIN: Can I just understand this. I'm starting to come back to this assessment. Are these...is this the EPA prescribed assessment...

THE WITNESS: Yes.

MR. LASKIN: ...and is it mandatory now in the States, or is it voluntary?

THE WITNESS: Well, as of this point the EPA part of it is voluntary. What has been done in order to increase compliance from the schools, is we have an agreement with the EPA that if the school follows this procedure, that if and when regulations do come out they will not be in double jeopardy, in other words, have to go through it all again. At this point

THE WITNESS: (cont'd.) each school has to pay for the analysis of the specimens, which is roughly twenty dollars per analysis for the asbestos, so even with tight budgets, ten or twelve specimens, schools boards don't like to spend that money needlessly.

So we do have that. We do have now new regulations out by the Department of Education, so we have bureaucratic problems of the Department of Education having come out with requirements that are somewhat different from what the EPA has proposed, and we are hoping that they will get together.

We are the recipient, at this point, of bureaucratic problems.

MR. LASKIN: If we go to the second last page of this little document, do I take it that if you...you inspect the building and there is less than one percent of asbestos, that's the end of the matter under these guidelines?

THE WITNESS: Generally, yes.

MR. LASKIN: If there is one percent or greater asbestos, you then apply this exposure factor score?

THE WITNESS: Yes.

MR. LASKIN: Do I take it that the control technique that applies then depends upon what score you get?

THE WITNESS: Not necessarily. What they have tried to do, and I don't think has been terribly successful, is people tend to equate a higher score with...well, then we have to remove it. That's not necessarily so.

You need to look at the situation, and if it's appropriate...it really means action is necessary, so that the higher the score, this means that you need to do something about it and then you don't apply a blanket thing...well, if it's over this, you have to remove it, when in fact it can probably be handled better by encapsulation. We have found, at least in the few we've gone to, the majority of cases there is less

THE WITNESS: (cont'd.) disruption, there is less risk of spreading asbestos around, and frequently they can encapsulate. But that's up to the contractor.

From our standpoint, we don't tell them what they must do, just that action must be taken. Then they go and, frequently, depending on how much money they have to spend, will choose which way to go.

MR. LASKIN: Your experience so far in the State of Wisconsin, is that you are not in all cases removing asbestos?

THE WITNESS: That's true.

MR. LASKIN: Have you developed any informal guidelines as to when you remove as opposed to when you might encapsulate?

THE WITNESS: We haven't gotten to that extent. Pretty generally we have tried to avoid cookbook-style evaluation.

In other words, it has to be done on a case-by-case basis and get in the contractors to say what can or cannot be done. They will say, look, this material, even if you encapsulate it, is just going to fall down, there's not enough adhesion to the underlying surface...or, you then get into building codes. If you remove it, it may not meet fire code any longer. So it's... all of this has to be taken into account when it's looked at, so I think the idea here with the score is to give you a ballpark sort of idea of which to apply some rational decision making.

MR. LASKIN: Have you developed any list of experienced or approved contractors who are giving out this kind of advice?

THE WITNESS: We have an informal list and we do have the EPA laboratory-tested approved list of sealants for encapsulation.

But generally we do not get into the role of recommending what contractor to go with. We just cannot do that. All the school boards have to put any job like this out on bids,

THE WITNESS: (cont'd.) so they really can't go with a sole source generally either, so we can put them in contact with who is in their area, because we do have a listing.

MR. LASKIN: Just for the record, Mr. Chairman, we should mark this bundle of documents as exhibit twenty-four.

DR. DUPRE: What number?

MR. LASKIN: Twenty-four.

EXHIBIT # 24: The abovementioned document was then produced and marked.

MR. HARDY: Shall we identify it, for the record?

MR. LASKIN: It would appear to be, Mr. Hardy, some materials which I take it, Dr. Anderson, were prepared by the EPA?

THE WITNESS: Well what it is, is it's part of the larger educational packet of documents that we distribute to the school boards, etc., and yes, I believe this chapter seven thing is coming from the EPA.

MR. BAZIN: Is that educational package part of the record? Exhibit twenty-four, chapter seven, Exposure Assessment, is part of an educational package?

THE WITNESS: Yes.

MS, KAHN: Yes, we'll make the rest of it...

MR. BAZIN: I'm not asking that it be part of the record, but if it is...

MR. LASKIN: Well, let's make it part of the record and let's make it exhibit twenty-four A, to differentiate it from what you have before. If the parties want it, we'll distribute copies of it.

EXHIBIT # 24 A: The abovementioned document was then produced and marked.

MR. LASKIN: Can I just come back to your household contact study for just a moment?

MR. LASKIN: (cont'd.) You have told us that you had no exposure level measurements. Did you make any attempt to try to simulate what conditions may have been in those households for the purpose of trying to get some rough estimate of what the levels of exposure might have been?

THE WITNESS: At one point I think Dr. Nicholson did some of that, but on a broad basis, no. I mean the homes were considerably different, and I would say in general, no.

I know there was some measurements, at one point, that he made, and I think that went into his risk assessment thing on the measurement levels that can occur.

MR. LASKIN: I see from the figures that you have given that there were a large number of people whom you didn't have in for examinations, and I'm just wondering how confident you are that the people you did examine are representative of the whole group of household contact workers.

I suppose what I think of is if I felt myself being a particularly healthy individual, I don't think I would want to come into your clinic and be x-rayed.

THE WITNESS: Well, that's always a problem. I guess the best way to answer that is what the purpose of the project was. It is not our intention to use this to extrapolate to all family members of asbestos workers or factories. This is a unique experience in this particular plant.

Now, you may be able to generalize to other situations, but that may not be the case. So by saying that we had thirty-five percent abnormal, we are not saying that we can confidently say that thirty-five percent of family members of anybody who worked with asbestos are going to have abnormal x-rays. So in that sense how representative the group is, is less important. In fact, I suppose it could go either way, that we could have underestimated and we could have overestimated, but as long as we are not...our main purpose was a descriptive

THE WITNESS: (cont'd.) study to see was there anything there.

Since it was different from the comparison group, whether the percentages go up a little or down a little is less important, and it would be unlikely that everybody who didn't come in had a normal x-ray. I guess the other way to look at it is, you see from the early figures of three hundred and twenty-six, the actual overall prevalences when you double a number of people that came in remain virtually the same. So I think we got a pretty good cross mix.

Part of the reason why I think we did as well, or why people were interested in coming in, is because this group of workers had a very, very bad mortality experience. So it was also a very close community and many of them knew each other, frequently were related, so that they all had known somebody who either has gotten compensation or has died of a lung cancer, or have a family member who has. So the level of concern in them was quite high.

Those that didn't come, primarily, are those out of state or who were already dead.

MR. LASKIN: Did you attempt to make any correlation between the household members whom you did examine and the worker, on the basis of the worker's illness or lack of illness?

In other words, was there any of that correlation?

THE WITNESS: We did look at that by looking at the chest x-rays or classifying whether or not the worker had an abnormal x-ray. Of course at the time we examined the workers, they were all many years from their exposure, and I think of those who were alive who came in, something like ninety-eight percent had abnormal x-rays. So that was not a very sensitive indicator.

So this is a group that by and large, as I say,

THE WITNESS: (cont'd.) those who remained alive are quite ill...except for the very short-term people.

MR. LASKIN: It wasn't a necessary prerequisite to examine the household member that the worker in the family be alive?

THE WITNESS: Oh, no. No. It just made it a whole lot easier to find that family. That's why the thing has gone on so many years, that it's extremely difficult to (1) locate who was in that household when the fellow who lived there has died.

Now we may know that in many cases he had family, because there is a wife next-of-kin listed, but the man may have come up from somewhere else and worked for six months and lived in a rooming house. So we can't include that family.

So the only family that we have included are those that we have been able to have a corroborative witness, as it were, a record that shows that that person did in fact live in that house at that time.

MR. LASKIN: In addition to the household contact exposure, was there any of what, for lack of a better term I may loosely call neighborhood exposure, in the sense...or any of the places that you were looking at, are they sufficiently close to the factory itself that there might have been some kind of exposure from the factory?

THE WITNESS: In some cases that's true. One of the things we did code was whether or not the house was within a half a mile of the plant. We haven't looked at that completely yet, but in the three twenty-six we did, and we did not see that as being a significant factor.

In other words, those who were family and within a half a mile weren't different from those that were family and not a half a mile.

Now, with the larger group we will do more.

MR. LASKIN: Is there any medical or biological

MR. LASKIN: (cont'd.) evidence to suggest that pleural changes have any relationship to the development of malignancies?

THE WITNESS: Well, that can go two ways, I guess. The easiest way to answer that is, there have been some reports by Dr....I think it's Burrow...out of following shipyard workers who had only x-ray evidence of pleural disease on their x-rays, no parenchymal abnormality...followed them over time to see what happened. They actually were very similar to the experience of shipyard workers. They did have excess lung cancers and mesotheliomas.

Now, you can't attribute that to the pleural change itself. Probably the pleural change represents an index that exposure has occurred, so that person independently is at risk, compared to something with nothing on his x-ray, but I am not aware that pleural plaques develop into mesothelioma. I think it's probably an independent sort of procedure.

I mean, some people with mesotheliomas have plaques, but also you have people...or pleural thickening...who develop mesotheliomas, that never had any previous evidence of it.

MR. LASKIN: I suppose what I'm struggling for.. I'm not..

THE WITNESS: I would say it's not a premalignant condition.

MR. LASKIN: I suppose in a general way, and I haven't put the question very well, but I suppose what I'm trying to find out is, if you take pleural changes alone, by themselves, with nothing else, what does the medical and biological evidence tell us about whether there are any adverse health effects down the line, from those pleural changes?

I mean, do they lead to something that is adverse?

THE WITNESS: Well, I believe...I would say ask that to Dr. Becklake when she comes, because she has shown that

THE WITNESS: (cont'd.) in pulmonary function the pleural changes are associated with decreased lung function. So in that sense...now whether that amount of lung function is clinically significant or not in an epidemiologic basis, it is associated. People who have severe pleural disease start with minimal pleural disease. Which are the ones that are going to progress and which are not, we don't know. But pleural disease can be incapacitating.

In many cases, like with these family members, it's more of a medical curiosity indicating that they were in fact exposed.

MR. LASKIN: Is there any effective treatment for these pleural manifestations?

THE WITNESS: For the severe disease, yes. They can do what they call a pleural decortication, which is...you strip off that pleura. You operate and take off the pleural thickening, which increases the volume inside the chest and it acts as...the pleura is inhibiting to the lungs expanding and contracting..or the chest wall too...so frequently people with advanced pleural disease will complain of, 'I have difficulty breathing'. The lungs that they have may be working fairly well, but the sensation is of their rib cage - 'I can't take a deep breath', because you have this constriction inside.

You remove that, they feel a lot better, but their lung function doesn't change remarkably.

So yes, you can take it out, but I would reserve that for extreme circumstances. I would not recommend ever that individuals who just have pleural thickening or pleural calcified plaques have them removed surgically. That is not indicated.

MR. LASKIN: Is the operation that you describe one that has a high success rate?

THE WITNESS: Well, I guess it depends on how

THE WITNESS: (cont'd.) you determine success. I would say..I have seen maybe five people that this has been done with, and the majority of them were better afterwards. But these were very sick people.

So it does prolong their life, but they are at a point where their life is going to be shortened already.

MR. LASKIN: From something I asked you before the break, I take it at this stage you can't tell us whether first of all there are any deaths in your cohort from asbestosis, directly or indirectly, and you are not yet able to say whether there is any excess risk of lung cancer?

THE WITNESS: No, I can't. I don't have that information at this point.

MR. LASKIN: Are you trying to suggest, or does one of the things your study suggests, that the risk of mesothelioma may well be a greater risk than either asbestosis or lung cancer, at low doses or short doses?

THE WITNESS: I don't think we can say that yet. The only thing we can say is, mesothelioma is very rare, and therefore when it occurs, even one case or two cases or three cases become significant. So at least in our population that we studied, there is indication that this group of people are at increased risk of mesothelioma.

Now if you consider that there's two thousand of those people and only four mesotheliomas that occurred, the absolute risk is certainly not extremely high.

Now, where lung cancer is going to occur more frequently, just in the general population, if there is an increase, clearly that could have a much more significant impact on total number of affected people. But clearly in this population there is an excess of mesotheliomas, and in general the association, as I showed you with the thirty-seven, and now

THE WITNESS: (cont'd.) I think it's much higher, there are many more reported cases. The problem is, we don't know...like, we'll be getting in this family...out of how many, what's the proportion of family members that develop mesothelioma from all these other things, because it's such a rare event in any case.

MR. LASKIN: Mr. Chairman, I note it's one o'clock and perhaps we should take the luncheon adjournment.

I won't be very much longer. I wouldn't think more than about half an hour, at most.

DR. DUPRE: The counsel for the other parties will have an opportunity to caucus to give me an idea of the subsequent timetable this afternoon, before we start at 2:15?

MR. LASKIN: Certainly.

DR. DUPRE: Shall we return then at quarter past two?

Thank you.

THE INQUIRY RECESSED

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THE INQUIRY RESUMED

DR. DUPRE: Counsel?

MR. LASKIN: Thank you, Mr. Chairman.

MR. LASKIN: Q. There was one comment you made just before lunch, in relation to your household contact study, and that was that it was...I think you used the word a 'unique' study. I meant to ask you in what sense you meant it as a unique study. In the sense that this was the workers themselves exposed for a very short period of time and that was somewhat unusual?

THE WITNESS: A. I guess more what I meant was that this was one instance that we can describe what we saw, from that plant. It was a plant, as I said, which made amosite asbestos

A. (cont'd.) products. There was no chrysotile used that we are aware of, so in that sense it was an unusual situation. So I think it's difficult to extrapolate, or what I want to avoid, as I said, is extrapolating from this one situation of workers and family members in this one plant, which was selected because it was nearby, we knew the workers had an unusually bad mortality experience, and I don't want to translate that into any other factory. I think it's probably fair to say that this has demonstrated that there is a risk that during the time these people worked there was not sufficient concern or protection to keep the dust in the plant, they took all their workclothes home. The women would describe how they laundered them and the dust that accummulated from that was sufficient in this instance to cause disease.

Whether it would be true in all others, I think you need to take it case by case.

Q. You lead me inevitably to ask you, because you made the point that only amosite was used, and we have heard a lot of evidence already about different fiber types and effects and so on. To your knowledge is there any parallel study or similar study of household contacts of an asbestos operation that used chrysotile asbestos?

A. I'm not aware of any where it could be documented that chrysotile was the only one. I have seen numerous individual cases of family members of insulators, for instance, and I am aware that right now in California there is a study of family members of long-term shipyard workers. But now they may well...and I'm sure they were...exposed to a combination of chrysotile and amosite and other fibers. It's very difficult...I'm not aware of any study which would have people exposed in the forties, whose only exposure would have been chrysotile, in a manufacturing situation.

But that individual cases can occur in insulators.

A. (cont'd.) We've seen them. It's just that it hasn't been a population-based type of study.

Q. When you say...you were careful to say manufacturing operation...do you also include user occupation?

A. Yes. Generally it's a whole lot easier to locate family members of people who are at a fixed site. I only say that because the insulators will frequently be on the road and will not be living at home, and they may not work with asbestos all year. They may be employed only part time, where the conditions in the plant tend to be relatively stable, rather than switching from job to job where it may vary tremendously.

Q. In the first article that you did in 1976, which is at tab two, if you turn to page 322, you raised a number of interesting questions which you seem to indicate required further study, and you hoped that your further studies might help clarify some of those issues.

Can I ask you generally whether your further studies have clarified any of those issues?

Are you with me? At...

A. Yes. I would say that we are beginning to get some information. As I said before, at this time I don't see that there is any strong correlation between the prevalence of pleural fibrosis and the subsequent occurrence of mesothelioma.

Q. What about the second matter you raised there? That's certainly of some interest to this Commission because of the possibility that children, people at young ages are exposed. Have your studies clarified or helped to any extent on that question?

A. At this point I think the only thing I can say is what...the one table I showed where the sons had a higher prevalence of abnormalities than the daughters. It couldn't be explained solely by duration of their potential exposure.

A. (cont'd.) There are many possible explanations, that the daughters stayed away or they did different things than the sons and therefore would have less likelihood to be exposed, or what. I don't know.

But at this point I don't think from our information that we can say either way, or I can say from our data on the family study that age of exposure made much difference.

In other words, we didn't see people who were exposed at infancy having a worse reaction or a higher prevalence than those exposed earlier. The only concern is that the same age discrepancy carried up to the point of exam, so somebody exposed at birth may have had abnormalities at age thirty, where somebody exposed at fifty, a wife, would have changes at age seventy or seventy-five.

Q. As a medical doctor, in your opinion is there anything in the makeup of lungs of young children that may make them more susceptible to this kind of exposure than an adult?

A. Well, there's lots of things going on in a newborn and thereafter as their lungs develop. I'm not aware of anything, or any studies that I could quote that would definitely say that a newborn infant is more susceptible to a dust disease than others. They certainly do have special problems and have different immune systems, but I don't think that we have sufficient information at this point to say what their special risk is, other than that they then have a whole lifetime of fiber residence in their lungs, which would have an added number of years at risk if duration of residence is important.

Q. What about these questions of the effect of the intensity of exposure on the one side, and the other matter you raise, the effect of interrupted versus continuous exposure?

A. I don't think from these studies that we can say much, other than that obviously these people did not have exposure for the years subsequent to this, and that they did have exposure for a relatively short period of time in the houehold, so therefore, as I said, more than half of them, the dust was brought into the home for a year or less. So this is a short period of time followed up many years later.

Unfortunately, we don't have the periodic exams to say when did the abnormalities first appear, to really know is there a progression or a dose relationship as to onset of disease. We only got them at one slice in time, and then distributed them over when they were exposed and how long.

But it was clearly there for a long time.

Q. Is one thing that your study suggested that the amount of exposure, at least in the terms that you measured it, which was index duration of employment, was inversely related to the latency period when these pleural changes manifested themselves?

A. Well, we can say that indirectly, yes, that the group exposed first in the 1950's had a lower prevalence of disease than those first exposed in the 1940's.

Now to make what you are asking, that assumption, we have to make the assumption that the dust brought home would have been similar and in similar amounts. All we have to go on there, of course, is description. As I said, after the war conditions may have changed.

But in any case, that group had a different prevalence which circumstantially would be attributable to a different time range. But again, you have to recognize the difference between a prospective study and a cross-sectional study, and this was a cross-sectional, and you are a little bit on shaky grounds to try to spread that out and assume that all of these people are acting as they would if you followed them prospectively.

Q. Can I just turn for a brief moment to the study of the maintenance workers in the chemical industry, which you dealt with and I think is at tab six?

A. Sure.

Q. You make the point...you and your coauthors make the point that maintenance workers are at special risk. I take it with respect to asbestos exposure. Can you elaborate as to what you mean by that?

A. I think our prime reasoning behind this is many of the maintenance men are unaware of their potential exposure, and here we are differentiating the general maintenance person who may do many, many different jobs in a given factory, and therefore he isn't taking off insulation on a regular basis as would a standard insulation man. Most of these people do it only on a piecemeal basis. In other words, there's a leak somewhere in a pipe joint, they have to go there, take off the old insulation sufficiently to repair or replace the joint, or a pump or whatever happens to be there. Somebody special isn't called in to take that off. So these men will go in and get at the job they need to do in as rapid a fashion as possible... don't think, 'is this an asbestos-containing material or is it not'.

Frequently they are dealing, as I say, with maintenance where there has been a problem, so that the integrity of the insulation is often already disrupted.

There may have been a long-term leak in the sealant, or whatever is on the insulation has broken down, and it's all loose and friable all over, as opposed to if you went in with a good, solid piece of insulation. So they are dealing with unusual situations.

Q. Do they tend to be relatively high exposure levels for short periods of time?

A. Well, that would be the presumption. Again,

A. (cont'd.) we did not go into these plants and make measurements, but clearly they didn't do this continuously during the day. All we have is, again, all of these are descriptive studies to see first, is there a potential problem, can we find biologic evidence of asbestos exposure, and that's basically what was done here. That if you have disease, other than for setting standards, it doesn't matter much whether it was a very low exposure or a very high exposure. Our job was to see, is there disease there, and is this an unsafe condition. Then subsequently you need to monitor that and see what changes can be made, and most of it can be handled with appropriate work practices.

Q. Do you know what the fiber type was that these people were exposed to?

A. I suspect again it was standard insulation, which would probably contain chrysotile. It may well also have contained amosite, but depending on the plant or the time, what product they purchased, this may change. So I would suspect this does not represent the pure exposure...or at least we have no way of knowing that that's the case.

Q. Was there any attempt, with respect to this study, to look at mortality?

A. No.

Q. I take it you can't..or can you help us one way or the other as to whether, for example, there were any mesothelioma deaths in this population of workers?

A. I don't know that, no.

Q. I just wanted to ask you a few questions about your...I'm sorry, go ahead...a few questions about mesothelioma, from the paper which at least you were a contributor to, although I gather you did not write, the book Asbestos and Disease.

A. Yeah.

Q. You discuss, at page 280 of that paper, the question of fiber type in relation to mesothelioma, and can I ask you whether the group of you who collaborated on this particular paper came to any conclusion as to whether there are different risks of mesothelioma, depending upon different fiber type?

A. Well, a lot of that depends on whether you look at animal data or you look at the human data. I think our baseline conclusion is that probably with the exception of anthophyllite, that chrysotile, crocidolite and amosite will cause mesotheliomas, or have been associated with them in occupational groups.

Now the prevalences in various groups has ranged, and that is where the discussion has come as to are some more mesothelioma inducing, prone, than others. I think by looking at the various studies it is very difficult...they are all done as the family study, almost unique to the group they are looking at, and it's hard to extrapolate from one to the other based on fiber count and size.

But as we have said here, on the basic strength there were differences and again from the animal work it is somewhat easier because you can control the dose a little bit better. Here, at least at the point I am quoting from that one paper of Berry and Wagner, it appeared that chrysotile, crocidolite, amosite were in that descending order.

But this is certainly one of the areas that is currently under discussion, and I think our bottom line was that mesothelioma does occur under exposure, occupational exposure, to these various ones and depending on the group, you get varying prevalences. There is probably ample means to explain those varying prevalences rather than to try to say is one much worse than the other.

But the different prevalences are there. You

A. (cont'd.) can't argue with that.

Q. Is that still, so far as you are aware, the prevailing position at Mount Sinai? Is that the prevailing view that it has?

A. I wouldn't want to speak for Mount Sinai in general. I can speak for myself and the discussions I have had at that time, but I do think that everybody there would agree that mesothelioma in humans is associated with all these various types of exposure.

Q. Don't make a distinction in terms of relative risk as amongst different fiber types?

A. Well, in terms of relative...you know, to say that there may be a difference, yes. That obviously there are observed differences in the various studies.

How meaningful this is or how one can translate that into some sort of policy or something, I think is very difficult. I guess...at least I tend to stay on the scientific aspect less than on the policy decision, and you look at whether or not it causes the tumor and it does in fact cause the tumor in the various populations, and it's very difficult, as I said, and there has been considerable effort to try to find human pure exposures, and that's...a negative is extremely difficult to prove.

Q. Is there any evidence of mesothelioma in the school system in Wisconsin, of which you are aware?

A. I don't believe so. We have...it depends what you mean by school system. As you may know by looking at the literature, there is such a thing as childhood mesothelioma, which probably is a different sort of thing than an asbestos thing. No, but I don't believe...I am not aware that there are any mesotheliomas that have been attributed to, say school asbestos exposure.

Obviously anybody who develops a mesothelioma has

A. (cont'd.) gone to school, and therefore you've got me in a little bit of a bind, because obviously I could say yes.

Q. Sure.

A. But I don't...I'm not aware of any cases where you could say that they had significant exposure in the school to warrant that as a causal agent.

Q. Just to come back to the point you made just before, is there a variant of mesothelioma which occurs in children, which is not attributable, or so far has not been linked to exposure to asbestos?

A. Well, it's a very, very rare tumor, and most of the effort has been to look at adults. I believe there have been a few childhood cases and I'm not that familiar with the specifics of those, but I believe childhood mesothelioma is very rare. Certainly it is not included in any of the environmental types of series we've seen.

Q. You also, at least the authors of this chapter make the point that there is evidence of mesothelioma in other parts of the world which, at least to date, has not been shown to be associated with asbestos exposure. I think you point out the rural community in India, and so on.

Is there any such evidence in the United States, of which you are aware?

A. I'm not aware that there is any clustering, say, of mesotheliomas that could be attributed to...or like the situation in India recently, reported by Dr. Sojoluchu that, you know, there is a community with a very high prevalence. No.

Really, as far as I'm aware the only area would be in the Arizona area.

Q. And what...

A. Well, where there would be...where there has been asbestos mining, or there is sufficient outcrops or asbestos

A. (cont'd.) in the soil that would be comparable to the India, or to the other areas.

Q. One or two other questions I wanted to ask you. In the paper...we are still in tab three, but the separate paper which deals with lung cancer. Beginning at page 309, the authors address the question of the causal relationship between lung cancer and asbestos exposure, and make...as I read it...one of the points that appears to be made here is that generally, although not always, ordinary lung cancer will appear in the upper portions of the lung, whereas asbestos-related lung cancer will appear in the lower portions of the lung.

How reliable is that?

A. Not very.

Q. Not very?

A. I would not say that, for instance in terms of compensating or something like this, that you could say if the tumor arises, it must arise in this area to be asbestos.

Statistically there is an appearance that it tends to be more peripheral and in the lower lobes, but that's a tendency, it's not a definite point.

Q. Is there some medical or biological explanation or support for it?

A. Well, I guess I don't know what the absolute explanation is. What I can tell you is what my understanding is, that most of the other types, or the smoking-related tumors, tend to be central tumors arising from the large bronchi, bronchogenic carcinoma..with the exception of some of the other cell types which are more peripheral. But that's the number one, the squamous cell is where it arises.

Now when similar tumors arise in the asbestos ones, they tend to be out further and more peripheral. Now why that is, whether there is some mechanism that has been possibly attributed, that fibers act as a carrier, and that's how these agents get out there or sit, there is really no firm evidence

A. (cont'd.) for all of the various speculation and unfortunately we really don't know why that is. It could be... it's like the fibrosis appears more in the lower levels, lower parts of the lungs, so therefore maybe this is an area that the asbestos makes more prone to the development of tumors.

Certainly it is a classic area for the fibrosis to appear, but whether there is any link between these two, because it makes logical sense doesn't mean it is scientifically or histologically defensible. I guess you would have to ask the pathologists.

Q. You lead me into the other question I wanted to ask you, and it's another matter you raise and we've heard some evidence on, and that is the relationship, if any, between lung cancer on the one hand and evidence of fibrosis on the other, and one of the propositions that has been variously put to us is that a dose of exposure sufficient to produce the carcinogenic response will also be a dose that produces fibrogenic response. I just wonder whether you have any professional opinion or judgement on that question?

A. As an opinion, I would say my feeling is that probably the carcinogenic dose is going to be lower than the fibrogenic dose. I guess I would base that on a variety of studies, like in the insulator studies or others, or the large number of cases I see where there is a carcinoma and there is no evidence of fibrosis.

Then again, you go the other way. Every case of fibrosis doesn't develop a malginancy, so I think there are probably two parallel track things. I think if you look at the various...if we want to call them latency period-type graphs, when you start to see excesses, generally you see excesses, the fibrosis appears earlier and then the malignancies appear later. The tendency is to think that well, it's these fibrosis people who are out here developing the tumors. That's not

A. (cont'd.) necessarily the case. It's very difficult when you have parallel things that are both dose related to be able to differentiate between the two activities, because they are going along and they are age associated too, and they are both related to dose and duration, so the only way to look at it is to get down where you have very low doses and no fibrosis appears.

Generally what's used as an indicator of significant exposure is the appearance of...like plaques or fibrosis on the chest x-rays. I'm not aware of studies done of asbestos workers who have perfectly normal x-rays to see whether their mortality experience is different from those...or from the general population...if they have had significant exposures.

So it's very difficult to separate those out. But I think there has been, at least in the patients that we've seen, the prevalence of fibrosis is going down. Like in the big insulator cohort...that includes everybody...of which very many have normal x-rays. Yet the only way we can sort out, they are all treated alike, they all seem to have the same risk.

Q. So that even with normal x-rays, they have got, you've found excess lung cancer?

A. Well, we haven't...we don't have that x-ray information.

Q. Oh, you are talking about...

A. I'm saying it's a whole group of which we know many do not have abnormal x-rays, but I have seen many cases of individuals with lung cancers, with normal x-rays.

Q. Who have been exposed to asbestos?

A. Who have been exposed to asbestos. When you get a biopsy they will have fibrosis, microscopic fibrosis, and they may have many, many fibers in their lungs, but if they are a short duration worker, a relatively short onset, they may

A. (cont'd.) not have developed a fibrosis yet.

Q. Are you seeing that phenomenon as well? Short-duration workers, less than ten years?

A. Yes, you see that too. There are fewer of those, but...because they tend to be younger. So there are many factors involved here, with age, that a young individual is unlikely to die from any cause and therefore the prime period of death is fifty-five on, and that's when you start to see these people, you start to see their chest x-rays too.

Q. Can I just finally come back to one questions on the schools document, which is exhibit twenty-four?

I just want to make sure that I understand the way you calculate this score for a school. The question I have really relates to factor number seven, which is asbestos content.

You may have dealt with this this morning and I just may have missed it, but how do you make that determination as to the percentage asbestos content?

A. Okay. That's done with optical microscopy, optical staining. You get a bulk sample, you reach up and get a little clump of something down, put it in a container and send it to the lab. They will take this out and generally dilute it with water and put it out and stain it.

There are a variety of stains that are picked up by the various asbestos, and rather reliably there will be different colours under polarized microscopy, and they would then...the person will look through the microscope and count the number of fibers that they see of the various types.

Q. It is a fiber count?

A. Yes.

Q. It's a fiber count percentage, it's not a weight percentage?

A. Oh, no. No, it's a very quick, inexpensive means to get a rough approximation. So you will see there it is like one to ten percent. Certainly there is...most specimens

A. (cont'd.) do not come into the lab in a uniform manner. There is all sorts of other things that have been used to put it up, so that's why that is used.

Q. Is it fibers greater than five microns in length, of greater than three-to-one aspect ratio?

A. It's a stain. There aren't...you aren't doing fiber sizing. Now obviously if it's too small, you can't see it. But they stain the fiber and it would be predominantly that, but it should not be compared to air measurements, for instance, of respirable particles.

What you are trying to do is determine what percent of this material is asbestos. You are not concerned if this is respirable particles or not, and you are identifying a mineral by its staining and polarized light characteristics. So that's how it's used.

DR. UFFEN: Can I pursue this a little bit, because I found this quite interesting as a procedure for inspecting schools.

But it's my nature to examine the sensitivity to data, I guess.

From what you just said in number seven, the Asbestos Content, if the measurement gave thirty-five percent, then it would be rated score three?

THE WITNESS: I gather, yes.

DR. UFFEN: Now, I went down to see what the worst possible situation would be, and in the process I think I got two things straight. I get two hundred and forty as the highest possible score. Do you know, is that...?

THE WITNESS: I really don't know. I haven't...

DR. UFFEN: I think if you give the worse score on each one, you come out to two hundred and forty. But in the process I was struck by the fact that items seven and eight, the asbestos content, which has just been raised,

DR. UFFEN: (cont'd.) and the next one, friability, are multiplied. Furthermore, they are multiplied twice. You multiply seven and eight together, and then you take them and you multiply the addition of the first six factors.

This would suggest that if you make a little error in the estimate, then that error will be just multiplied quite a bit.

Have you had any experience yet as to how big this potential error could be?

THE WITNESS: Well, one, the error obviously can be very great because you are taking one little tiny sample off the corner of the wall, which is said to be representative.

Now if you then go back in and take it from this corner over here, which is perhaps different, it could vary.

I guess at least the way...and I don't want to defend this system because it's not my system, it was devised, as I say, by EPA, and why they give the weighting they do, you would need to ask them...but the way we use it is strictly as an indicator, in that we don't give any greater weight to a much higher number or a middle number. It's more a means to assess, something needs to be done.

This is, I guess, a compromise, because people like to have a number.

Now generally you can go in...and that's why it is multiplied...the really important things are, how likely is this to get into the air, how friable. You look up there, if it's obviously falling down because you can see clumps of it on the floor and there's foot tracks out of the room, and it's a hundred percent asbestos, you are more concerned than if sixty percent of it or a hundred and ninety-eight percent of it is plaster of paris, which would look very similar.

In a number of the schools there has been a lot

THE WITNESS: (cont'd.) of concern because this stuff is falling down, and you look at it and it doesn't have any asbestos in it. It's another type of sealing material.

DR. UFFEN: Could I pursue this just a little bit? One of the things you have to be worried...or I would be worried about, is giving a false appearance of precision inadvertently.

So I did a little exercise, and I'll put it to you and see whether my understanding is correct. I assume there would be two inspectors and they go in and they get the same assessment, and on items one to six I assumed they both got a total score of eight, but on items seven and eight, one of them scored three and the other scored four, and one of them scored two and the other scored three. Just one little difference in the score.

When I worked out the numbers, the exposure number went from forty-eight to ninety-six. Now, forty-eight would be below the criteria of fifty, of significance, whereas I would think ninety-six would be regarded as a huge number.

All of this led me to believe that what appeared to be a good procedure had a very considerable factor of...

THE WITNESS: Well, yes, you are right. You have to apply...any scale like this has to be applied judicially. It cannot be...and these, as I say, are all guideline things...it's to give you a ballpark idea of where you are and what you ought to be doing, not a concrete thing.

As far as the likelihood of having that great an error in the percent of asbestos, if you take the same sample and send it around to various interpreters, they will do very well. They do have, EPA has a standard set of blind specimens that come through, that the lab does, and they are generally certainly within the variability within the groups.

Now, it's true that you could go from a ten percent to an eleven percent, and throw you into another category. Anytime that you have a scale like that, you have to look at that. I would

THE WITNESS: (cont'd.) hope before one would take action you would look to see what brought this on, and of course the laboratory technicians are also going to look at that specimen, count it a number of times, and will make a concerted effort to see does it really belong in a higher group.

I mean, there's a lot of subjective decision making when you come to 'what are we going to do about it', and you will take that into account.

DR. UFFEN: Could I ask just one more question about the form? I'm looking at this one now. It doesn't have all these little caveats on it, and I am an anti-bureaucrat and when I can see somebody get a hundred of these things to fill out, and they get down to the bottom here and the exposure number takes on a significance that it should never, ever...

I also see, however, that there is a little compartment for air sampling - sample number, volume and results, and fibers per c.c. Did I understand you earlier, though, to say quite emphatically that fiber counts in air monitoring is useless?

THE WITNESS: In a school setting. You see this is a form, a standard form...anybody in the State of Wisconsin who comes in with their twenty dollars can have a sample analyzed, so this is a service that is also utilized by some industries. Sometimes air samples may have been taken, and under some circumstances would be warranted.

In general in our school...and I would say...not in general, flatly, we do not recommend air sampling in the school.

DR. UFFEN: That's a little hard to...I won't belabour it, but at the top of the page it says School District Number, and it would lead you to believe that this was intended for schools.

THE WITNESS: Well, it was adopted, as is every other form that has ever come along out of any government. It

THE WITNESS: (contd.) went from one to the other.

But yes. But we do not have air samples done in the schools. I mean we don't recommend it. Some of them may do it, but we do not recommend it.

MR. LASKIN: I have no more questions, Mr. Commissioner.

Thanks very much, Dr. Anderson. My friends here behind me and beside me may have some questions.

DR. DUPRE: Just before I turn to the parties, since we are on the school thing, can I just...

THE WITNESS: Sure.

DR. DUPRE: ...ask you about the administrative geography, what the situation is. Which state agency in Wisconsin is responsible for the asbestos identification and control program in the schools? Is the State Division of Health?

THE WITNESS: The Division of Health has been appointed by the Department of Education as the lead agency. The collection is done by the field staff of the Department of Industry, Labour and Human Relations, and we maintain the State Lab of Hygiene which does the laboratory analyses. So that's partly why it was an administrative decision on the part of the governor to give it to us.

DR. DUPRE: Once your lab has identified asbestos levels, and at this point a control program is going to go forward... whether it's removal, encapsulation, enclosure...is it your division that still plays the lead role as far as the state is concerned, or is it the Division of Education that comes into play?

THE WITNESS: With us, the monitoring or the data collection role is ours. Now, we also happen to have as part of this program a research project funded by NIOSH, so that one of our concerns was the followup to see...you go in, you do this program, is anything ever done by it. That's what our research

THE WITNESS: (cont'd.) project is doing, is this followup to see...now you've gotten, you've collected your samples, you've sent the results back to the school board with a recommendation, and what have they done. Because we have no enforcement.

So now our job is to go back in and see.

DR. DUPRE: So it is then entirely up to the school board whether or not to take action, and if so, what kind of action?

THE WITNESS: That is correct. That is correct.

DR. DUPRE: And the State Department of Education, or your Health Division, does not have anything to do with it?

THE WITNESS: No, not as to whether action ought to be taken.

Now, under the federal education rules, schools must be inspected. So the school board must send in samples, and part of the difference between that ruling and EPA is a factor of three. Education said you have to do three samples from each site, which of course triples the cost. EPA was saying you only needed to do one sample, so they wanted...I mean the question about the numbers that if you have an overzealous inspector, he could pick an area that - boy, that's got a lot of asbestos, I'm going to take that, and it isn't representative.

So they wanted three different sites and then you'll average them, because that will give you less, hopefully, variability. Again, it triples the cost.

DR. DUPRE: Now at present are there either state or federal funds that are made available to school boards that pursue a control program?

THE WITNESS: No.

DR. DUPRE: May I ask the parties for the batting lineup? Who is leading off? Mr. McNamee will lead off?

DR. DUPRE: (cont'd.) May I just ask who will bat second?

MR. BAZIN: We haven't worked it out yet.

DR. DUPRE: You haven't worked it out yet?

MR. BAZIN: No problem.

DR. DUPRE: No problem? All right, Mr. McNamee, lead off.

CROSS-EXAMINATION BY MR. McNAMEE

Q. Yes, Doctor. Dr. Dupre just asked his last question that I was going to ask you, because when Dr. Nicholson was here, one of his briefs, or was it tab ten, he appeared before the Oversight Commission. Are you aware of his testimony before that?

A. I know he did. I...

Q. In 1979, and...

A. I can't tell you what he said, exactly.

Q. One of the representatives from the House of Congress, I guess, a Mr. Miller, indicated that this was a situation crying out for some kind of federal help, and he mentioned that they had spent three hundred million dollars on six cases of swine flu fever. He thought this was somewhat more serious.

So you haven't had any federal response or federal funding yet?

A. Oh, we've had a response. Our budget has been cut. There are no new programs coming out of Washington. That's a state perspective.

Q. Do I understand that you have received responses from fifteen hundred schools out of twenty-three hundred, approximately?

A. Yeah. I think that was it. I don't have my thing here. Right.

Q. Maybe I missed the figures, did you indicate how many of those fifteen hundred schools contained asbestos?

A. We don't have that yet. All we've done is we have...see, a lot of the schools started the program before the data collection system was in place. We set up the system to do the testing for them, but there was no mechanism or dollars to computerize or do any kind of data collection. It was just coming in and going out, and that was part of our reason we felt that that was inappropriate, and NIOSH did too, so they gave us some money and we are sort of catching up right now.

But no, I can't tell you what proportion of the schools. Right now we are just trying to get each school inspected at least once.

Q. Did you indicate...I believe you used the figure seventy-five, having demonstrated some kind of compliance?

A. Yeah. We've gone back now...I can't tell you exactly what we used. We gave them three to six months after the first inspection. We then went back, and I think it was primarily first to those with...where recommendations to do something were made, and we went back to a hundred and twelve.

So of that hundred and twelve, seventy-five had in fact complied with the recommendation and had in fact done what they were supposed to. And that may have ranged from removal to encapsulation to just putting a little tag on a pipe in the basement.

But in any case, somebody went through and actually followed up on the recommendations at the school level.

Q. Have you actually done any either removals or encapsulations of complete schools?

A. We haven't done any of that.

Q. You wouldn't have an idea of the kind of cost?

A. No, I couldn't give you that figure at this point. I know there have been some that have gone into it in a

A. (cont'd.) big way, and then others, it's kind of run the whole gamut.

But in general I would say that the budgets have not been massive to try to alleviate the most pressing concerns.

Q. Well, Dr. Nicholson gave some evidence about his examination of New Jersey schools. Are you aware of his papers on that? Maybe he did two or three papers.

A. Some of the measurements there, yes. I couldn't quote exact numbers, but I'm familiar with them. Yes.

Q. Following his evidence I followed up a little bit, and although it isn't evidence, I checked with certain people in New Jersey and I talked to them on the telephone, and as I say, it's not evidence...a Mr. Richard Gates, the architectural supervisor, and I just threw two or three questions at him. One was the question of encapsulation versus removal. He indicated to me that New Jersey has ruled out encapsulation because...on two grounds. Basically when you encapsulate and you consequently have to do roof or ceiling repairs, you wind up with a subsequent problem, so they've gone...if there is friable asbestos present that is perceived to be a danger, they go for complete removal.

Are you aware of this?

A. I'm not sure what the official statement is there, no. I don't know.

Q. What about the observation that when you encapsulate and subsequently, say, you have roof or ceiling repairs, you might have a problem?

A. Oh, I think that's true. I think the potential...obviously the best answer is removal. Once the stuff is gone, obviously it can't cause you a problem there. If it's inappropriately removed, it could be a problem.

What we've found...again, I think our experience has been on a very practical level...that's very costly. To go

A. (cont'd.) in and remove asbestos from large areas both takes a lot of time and is very costly. Where we can't provide the funds to do either, in general you can contain the problem and of course the whole issue then, and what we tell them all is, you must also maintain maintenance and an awareness.

In other words, the problem for the time being, as long as the encapsulation is effective and is routinely inspected and found to be okay, you are safe. But obviously down the road, if there is a hurricane and everything gets blown around, or there is a fire in the building, or a major renovation is required, this will need to be identified and treated accordingly.

You are buying time with encapsulation, is what you are doing.

Q. All right, now...

A. And that becomes an almost socio-political decision as to, do you want to handle the problem in this manner today and recognize that you have only partially dealt with the issue.

Q. Dr. Nicholson indicated that...I think he had examined forty-eight schools in New Jersey, and thirty-three showed visible damage from erosion, pupil mischief and things like that. He also sort of subcategorized that in saying that there appeared to be greater damage from asbestos installations consisting of fibrous spray material versus the cementitious asbestos spray type of installation.

I asked him a couple of questions about whether it is possible, considering that some of the installations have been in there for maybe thirty years and upwards, whether any meaningful studies could be done. He indicated that studies could be done, they would be costly, and I don't think he indicated whether they would be meaningful studies.

I am just wondering whether you, yourself, would

Q. (cont'd.) consider that any meaningful studies of asbestos-related diseases in school children could be done, say with cohorts of thirty years? Say the first exposure was thirty years previous in a school containing asbestos.

A. I guess I would agree with the first part. It would be very costly.

I would suspect it would be possible to do something that would be meaningful. If you ask me could I do it or do I know where it could be done, I would have to say no.

As I say, the problem of following up school children over that period of time, all of which will go out into the work force and get their exposures in a variety of other ways incidental to the school, I don't know. I think it would be very difficult to do and since the exposed population is potentially so large, even a very small risk would be considered significant.

If, say, as opposed to a smaller population, you might be willing to accept a lower...or a higher prevalence of disease, therefore you would have to examine a great number of children. And I'm talking in terms of taking, say chest x-rays, because you would have to follow the others for mortality. Even if they are exposed thirty years ago, they are only going to be in their forties and you are going to have a very small number who died, and the majority of those are going to be auto accidents and suicides.

So you just...they will not have gotten to the age where you are going to have sufficient mortality to start to be able to see a difference from background.

Q. In one of your papers, I think it was at tab three, you did indicate that with mesothelioma you could have these trigger doses that might be actually quite small, and you could have a visible reaction say three or four years, that you would actually have a case. You wouldn't think this would

Q. (cont'd.) show up? You could find any significant figures on a past study that...

A. On mesothelioma? I think it would be difficult. Again, that's something that is so rare that to define a cohort...I guess...you know, I'm not saying it can't be done or it shouldn't be done, but I would think before embarking on something like that you would have to be extremely careful. This is the sort of thing you are only going to do once.

Q. Would I be wrong in suggesting that thirty years from now you are going to run into the same type of problem so that really to get any significant results of the school studies may very well be impossible. You just have to extrapolate from other data?

A. Yeah. I guess from my perspective I'm not sure that what we need to have is that you need to document that actual disease has occurred. What we are talking about is that there is a potential hazard, a health risk, there. And therefore we ought to take action.

You see, as I say, the ceiling falling down. I'm not sure that, you know, we need to get to the point where we say, aha! see, I was right! We've got X number of deaths.

What I would like to think is, we just go ahead and recognize this as a hazardous situation. How hazardous? Is it an imminent hazard that..like some of the schools have done where you evacuate everybody the day we hear about it, or is it a hazard that needs to be dealt with in a rational, planned sort of manner.

I think that the research community may, if there is sufficient interest that funds are available to do this, you may get some requests to do such a study. And I'm not saying don't do it, but I'm saying I wouldn't wait to take action until we have a positive study.

MR. McNAMEE: Thank you, Doctor. Those are my questions.

DR. DUPRE: Thank you, counsel.
Who wishes to bat second?
Miss Jolley?

CROSS-EXAMINATION BY MISS JOLLEY

Q. I have a couple of questions, because John covered a number of the areas that I was anxious about.

There was a statement on page 262 of tab three, which just was a little startling to me. That was, that asbestos was sometimes injected surgically into the pleural cavity, in the 1940's, to promote the formation of adhesions.

A. That's right.

Q. Was that a widespread...?

A. How widespread, I don't know. As this whole book, it's really an historical thing and it's going through virtually all the literature that we could get available and reporting what was there.

But, yes, it was used...as talc was used, too... to cause pleural adhesions. There has been some interest in trying to go through hospital records to identify such individuals and follow them up, but again that has been something that there has been talk about doing, and the actuality of identifying these people is very difficult.

Q. But it hasn't been followed up?

A. No.

Q. Okay. I just found it quite startling, human experimentation.

The other...one question just as a knowledge question. Geoffrey Berry was before this Commission and mentioned a mesodermomas. He didn't quite know what they were, and I wondered if you did. Mesodermomas. I'm not sure if I'm pronouncing it correctly.

A. I'm not sure. I could hazard a guess. That might be what is referred to as benign mesothelioma, as opposed to

A. (cont'd.) malignant mesothelioma. But these are things that are operated on, operable, can be removed and the patient successfully recovers.

I don't know...I would hazard that that might be what he is talking about.

Q. The one question, just following from a discussion that you had with Mr. Laskin about lung cancer, the causal relationship between asbestos and lung cancer, for compensation purposes one would not have to have fibrosis of lung in order to suggest that the cause was asbestos. That's your finding?

A. You mean for a tumor?

Q. Yes.

A. Yes. I would agree that fibrosis is not necessary to be present, and I think asbestos exposure carries with it the risk of developing malignancy, or an increased risk, and you don't necessarily have to have both of these present.

I would, however, be surprised if you did get lung tissue, if it was an asbestos-exposed individual with a tumor, that if you got a lung biopsy I suspect you would see numerous asbestos bodies and probably some microscopic fibrosis.

I think what's an important point in relation to this, to make, is that we are sort of...because the people you are getting in here are not pathologists, and we are speaking in terms of, as though we are making a pathologic diagnosis. I think it's important to know that what we see clinically is really pretty crude. By the time you see something on an x-ray..we've got trouble seeing a bullet on x-ray sometimes, and, you know, that's a pretty big thing, so for the fibrosis that you see under a microscope to get thick enough so it casts a shadow on an x-ray, you have to have quite a bit of these little guys in the lungs, and it takes awhile to get there. The same with the pleura. You'll see in the literature the pathologists saying, I

A. (cont'd.) see pleural plaques all the time.. when they go in at autopsy.

But the question is, how extensive are they and have they appeared on the x-ray. So what we are describing here is a different entity, that in one sense is almost an end stage entity. It's not the early changes. X-ray changes are not an early sign.

Tissue changes are much more reliable and perhaps even more nonspecific in early changes, and that's the sort of tissue that we don't have that much of...of the man who has worked three to five years and is killed in an auto accident, and an autopsy is done. Usually tissue is not available, so the only time we get them is either after the man has died, where it's too late to talk about early changes. Right now I would say if you are looking at the field where physicians are going as towards early diagnosis and predictive things...I think Dr. Becklake will probably be able to address those issues...what does it mean to see x-ray changes in a thirty year old man who has worked ten years? Is that worse or better than a fifty-five year old man after thirty years of work having the same appearance? Rather than other questions.

Q. Just following on that discussion, in tab four, page 2499, you make a recommendation that the study does... just at the end, the very end...that the study does suggest, however, that individuals found to have pleural thickening or calcification unrelated to infection or trauma should be followed closely.

What does that mean?

A. Well, I think that this indicates a potential risk. At this point I think we are tying to determine what that risk is. That when you see somebody like this for the first time, if you haven't been following them serially, you don't know. I mean you may just be picking up an early

A. (cont'd.) malignancy, because an early mesothelioma can look just like pleural thickening. The thing that really differentiated is that three months later the mesothelioma has filled the chest and the benign pleural disease is still there. So it becomes for the clinician extremely important to know what the previous x-ray was, and in my experience most or a lot of the patients aren't aware, even if it's benign, what's there, because the physicians are concerned if I tell you you've got a scar on your x-ray, you are going to get worried.

Well, now, he comes...he's on vacation fishing up here in Ontario and gets a chest pain, goes into the hospital, has an x-ray and here is this horrible big scallopy thing and they say, gee, I'm sorry, you may have a tumor...did you ever have anything on your x-ray before?

Not aware of it.

It could be that it is pleural thickening that has been there a long time, so the whole thing is to follow it over time. If there is very little change, it is very unlikely to be a malignancy. But I think it also...especially if the person has been occupationally exposed, this indicates that he certainly has had enough exposure to create a biologic response that we can detect on the chest x-ray.

And that puts him...if he is a cigarette smoker, I tell him to stop. It may be awhile before we have the information on what the meaning of pleural plaques in relation to cigarette smoking and subsequent disease, but I think it's just plain prudent for the treating physician...and I separate that from the epidemiologist hat, I'm switching off here.

When you've got a guy in front of you, you have to take the prudent action, which is to say, look, this indicates you have been exposed, it's important for you, amongst other reasons, this is one more reason to stop cigarette smoking.

A. (cont'd.) That's our biggest educational push right now, is to convince these people of that. Of course even if the asbestos turns out to be totally benign, you've done them much good if they stop smoking.

Q. Do you have any recommendations as a physician as to exactly what the medical surveillance of asbestos workers should be, how often and what?

A. That, again, is I think an issue that is being looked at. I think what's currently in the asbestos legislation... of periodic medical exams, chest x-rays, I'm not sure, that certainly is fine for detecting fibrosis, which is usually slow onset anyway. Whether this is going to do a great deal to assist in the early diagnosis and successful treatment of cancers, I'm not sure.

I guess, especially now that I'm officially a public health bureaucrat, I think prevention is the name of the game, not diagnosis and treatment.

Now we have a whole lot of people out there that need diagnosis and treatment, so you can't ignore that. I think it's important to get in and be seen by a physician, be it for blood pressure or asbestos disease. But that periodic exam isn't going to be what stops the guy from getting his disease.

I guess I would say a chest x-ray, a periodic physical...right now I think there is additional work needed in assessing the usefulness of sputum cytology. There are pros and cons on that right now.

It certainly is...

Q. That was my next question.

A. Well, there are certainly a variety of studies that are underway, all of which have problems with them, not the least of which is compliance on the part of the participant.

It's often true of any surveillance program, it's very difficult to come in on a repeated basis, voluntarily.

A. (cont'd.) We have that more of a problem, I would gather, in the States than you do here...at least what I'm familiar with, getting a miner's medical and certification. You don't work if you don't get your exam.

We really don't have that sort of a system. It may come that we need to do that sort of thing.

But I think cytology needs a real hard look. It has potential usefulness, but it needs to have a lot more rigorous science applied to it. I don't think there is anything that we should ever delude the workers into thinking that there is a panacea, that you go in and if you come out of it with a clean bill of health that your risk is gone - now I can go out and do whatever I want to do, because we just don't have that sort of technique. But there are a variety of other research things that need to be looked at.

But I would say the prime thing - you get them in for a periodic exam, you must have a good education program. You've got to get at them all the time for the smoking, for good work practices...it's so easy to get sloppy.

Q. I have just a couple of other questions.

One of the questions I have flows from tab seven, and it was a statement you made also about the maintenance workers in the chemical industry, and that was on page 397 of tab seven.

You were taking a subgroup out of the family cohort, and those were the people that actually had occupational exposure to asbestos.

A. Mmm-hmm.

Q. You said in that, which I found again, it was interesting, that nineteen...only nineteen people knew they had occupational exposure, and that was nineteen out of, I think, fifty-one. That is a very low number that actually knew that they had been exposed, and I think that you mentioned that a lot of the other maintenance workers were not aware. Is that

Q. (cntd.) general in the industry, that the workers in maintenance are not aware of the fact that they are being exposed to that?

A. I don't think it's fair to generalize to every industry. Certainly in the case of these people, if they are typical of what walked in for some other reason, they at least reported to us they were unaware.

It's very difficult from a physician's standpoint, and I've been at it both ways, of having examined somebody five years ago, have them come in and say, were you ever told you had an abnormal x-ray, and I got the letter I sent them, and he says no, I've never been told.

So what is retained and what the facts are can change. I would say that there is probably many people doing general maintenance that are unaware that they may work with asbestos. I would hope to think that that is improving, but I cannot say that I've done a survey, and I certainly wouldn't want to say that this is general, in any manner.

I would ask the workers.

Q. We are trying to do something.

Just taking up the maintenance workers at schools, a number of people, in the discussion of public buildings, clearly the priority must be set on the removal or dealing with asbestos in the public areas where there is a lot of exposure to children and where large numbers are going to be exposed.

A number of people, yourself included, have sort of said, if it's in a maintenance area you don't...that's not a a high priority. I'm a little worried about our maintenance workers in schools, because we have had a number of maintenance workers in our school system in Ontario have mesothelioma, etc.

But that's not to say that we shouldn't also deal with that problem?

A. Oh, I'm not saying that at all. I guess what

A. (cont'd.) I'm saying is that I think when you have a small group of people who are going to be having intimate contact with a given area, you can educate them and alert them to the problem. What we do, or part of the recommendations, is to label that.

In other words, they are to take a little tag that says 'this material contains asbestos, handle with care', wrap it around the pipe, twist the lock, and somebody comes by, because there is a leak, later on. Hopefully they will see it and if they are properly educated will say, hey, I better find out what to do here.

Now, that may be wishful thinking, but in those areas I don't think it's realistic to expect that we are going to redo all of the power or heating plants in every school. You can certainly restrict access to the area, and be sure that the guy who goes in there is properly trained.

I don't think...I think it is important to stress that education and training...I think asbestos can be handled safely. It's a dangerous substance, yes, but it can be handled safely when we know how to handle it safely, we know how to remove it safely.

It's a matter of putting that into practice and getting the person educated, and I think that's what we are getting down the line now, out into the field, to the maintenance people, now that, you know, studies like this one have said here's a group of people that seem to have sufficient exposure to have caused x-ray changes, you better alert your men.

Once alerted, a lot of practices can change. But if you don't know it's there, that's, I think, part of the problem in the schools.

Q. Our concern is, too, that some of the maintenance people have their offices in boiler rooms, and that kind of thing, and eat their lunch there, etc.

Q. (cont'd.) Just picking up on the public buildings, you mentioned the fact that you have no control over the contractors in the sense of how they are going to do it. Is there any state in the U.S., or any jurisdiction where you know where contractors removing asbestos are licensed, or perhaps controlled in some manner?

A. I just don't know. It may be, but I don't know. I suspect contractors are licensed under various procedures.

Now, if for instance, they have more than ten employees, then in the States they should come under the OSHA regulation and inspection. I'm the wrong person to ask... actually, I'm the wrong person on the asbestos control program, too. I happen to be interested and the nominal medical head of it, but the nuts and bolts of how it works, I don't really get involved in, and the same here. I would suspect you could contact somebody at NIOSH or EPA.

Q. Right. Then the next question was just the question...you said the removal costs are so great, but the costs of maintaining inspection and the ultimate removal will ultimately put encapsulation costs up fairly high as well, won't it? It would add down the line?

A. Oh, absolutely. It's a matter of when do you want to spend your money. The second question is, when do you have your money.

Q. Right.

A. If it's a matter of...at least...I think you have to take a very practical approach to the thing. If you can provide funding, then you can say you must do something, or at least low-interest loans, or something. That's what has been talked from the federal government.

But if, for instance on our level, at the State, if we go in, what we want to do is we want to do the best we can

A. (cont'd.) to eliminate that hazard.

If we can do it by the best possible means, fine, that's what we go for. We don't recommend, well, do the cheapest thing. We recommend do the best possible with what you have to work with.

That's why encapsulation, I think, becomes at least in the short term, a viable thing. It may not turn out in the long run to be the proper decision, and that I think is, you know, that becomes a social issue. If the money is there to handle that situation, obviously the sooner you get it out, the better.

MISS JOLLEY: Thank you, very much.

DR. DUPRE: Is this an appropriate moment to take a brief break?

MR. LASKIN: It is.

DR. DUPRE: Fine. We'll reconvene at five minutes to four.

THE INQUIRY RECESSED

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THE INQUIRY RESUMED

DR. DUPRE: Dr. Anderson, are you ready?

THE WITNESS: Oh, I'm ready, sure.

CROSS-EXAMINATION BY MR. HARDY

Q. Dr. Anderson, in discussing the family contact study which you did, at one point earlier today you referred to the condition in the homes of the workers as a condition of active contamination. I wonder if you could describe to us your understanding of how asbestos got into the home and to what extent it would have remained?

A. Okay. What I mean active, that's in terms of the definition of our cohort, which means we assumed that the

A. (cont'd.) mechanism that the amosite asbestos got into the home was via the worker. Now this came partially from circumstantial evidence of them reporting it, as well as Dr. Nicholson went into the homes and gathered settled dust. That's one of the benefits of having the amosite asbestos as opposed to chrysotile - there are very few home products that have amosite asbestos in them and therefore he felt fairly confident that if you found amosite, it had a high degree of probability of coming from this plant.

We didn't go so far as to try and tag it and see if in fact it was where it was from, but that in essence is how we assumed it came in...probably on workclothes, on shoes, in the automobiles and workclothes that these people may have worn and that the men would describe, one of their basic anecdotal stories was that this was a mixed population of blacks and whites, that you could tell who was who when you went in in the morning, and when you came out at night, everybody looked the same.

Q. White, that is?

A. White. Unlike the coal mines where it would be the other way around.

Whether or not, of course, this was the asbestos or the other calcine product that was added to it, of course, we don't know. But in any case they described very dusty conditions that they would bring home, and this was the understanding of how the asbestos got there.

As I say we...I say 'we' collectively, but Dr. Nicholson did go into a number of these homes and was able to find in the settled dust in rafters and in the basement, amosite asbestos some thirty years after.

There has also been a variety of studies, not in households but other types of circumstances, showing how the very fine, small fibers can remain entrained in the air and circulate quite readily. The very light ones can remain suspended for very

A. (cont'd.) long times, and therefore it's one of the things we are trying to look at is whether just residents in that home, whether it was a dropoff or not. And getting back to the first part of your question, the active contamination, we considered the period when the worker was actively working and might well have been each day bringing home his workclothes, as opposed to when he went to work somewhere else.

It doesn't mean the continued exposure in the home wasn't active. He just wasn't bringing in new material.

Q. So then I gather that contamination in the home with amosite fibers could have continued after the point where the workers stopped working in the amosite plant?

A. The contamination would have remained in the home, yes. That's I guess, different between bringing in a new supply. That's the differentiation we made, was that certainly it was still there after many years, so it is a possibility that everytime you vacuumed or dusted, you would reentrain the material.

But at this point we felt, and it would be best to correlate it with the actual worker time, because we assumed that many doses would be gotten by the housewife who would shake out these clothes, or the worker, the family members that would be around at that time.

Again, the actual exposure doses are conjecture.

Q. You just mentioned, I know, in your article at some point you discussed the laundering of the workclothes by some members of the household.

A. Yes.

Q. Do you have any further description you could give us as to what that process involved and what sort of exposures might have occurred?

A. Again, there, of course, was no standard procedure other than they used soap and water. But generally what would happen, as it was described to us by both the workers and

A. (cont'd.) especially the wives, they would first shake the clothes out to get the majority of the dust off, then depending on the earlier years, many of them wouldn't have electric washing machines and so it would be a hand washing machine, hand wringer, and then external drying outside. Some of them, as they came on the market, would have gas or electric tumbler dryers, so that's why I say it varied, and at this point we have not looked to see whether those who had one type had a different experience than the others. But that is a possibility to look at.

Q. One type of study that has been done, I know, by others, including Whitwell, of mesothelioma victims, has been to determine the number of fibers in the lungs at autopsy, and compare those fiber counts, I guess, with control groups.

A. Mmm-hmm.

Q. Have you done any such work with mesothelioma, case histories, related to this amosite factory, either with workers or family contacts?

A. We haven't been able to on the family workers. At this point we do have some tissue on I think two of the mesotheliomas, but the majority...I shouldn't say the majority, I believe all except one did not have an autopsy at death, and the only thing we have are surgical biopsy specimens which were primarily of the tumor itself.

We have tried to...and that's one of the things, part of the mortality study now is to gather what autopsy material is available on those who have deceased and go through the same procedures with the insulators, trying to get the best available evidence there, which is why I can't answer 'did the people die of asbestosis or not'. At this point the only information we have is what is on the death certificates, and we are now getting the medical records.

But we hope to be able to look...it would be very

A. (cont'd.) nice to be able to...if we got sufficient lung samples...to look at the dust on those lungs.

Again, what happens to the fibers, how many are dissolved or otherwise expelled, we don't know. But it would still be very interesting. The feasibility of it is the big problem.

Q. Will those sorts of fiber counts in the lungs be useful in attempting through another means to determine exposure levels of the family contacts?

A. Of what the exposure levels might have been?

Q. Right.

A. I don't think so. I don't think we can equate how many fibers you see to how many fibers were in the air, that there are so many...the vast majority of fibers never lodge in the lung anyway. They are exhaled and they may be excreted, so I guess it would only be interesting from the standpoint of, do they also after all these years still retain fiber in their lungs and possibly actively working.

But I don't think that by knowing that this lady has...you know...X grams per lung, or X number of fibers per lung is going to translatable into five years at two fibers or five years at one fiber. I just don't...I have never seen that that has been a practical sort of activity.

Q. It might...might not it, however, give some correlation to the amounts of exposures the various industrial workers will be...groups got over the years, at least on a relative basis, perhaps?

A. I think that would be somewhat dangerous. I think, yeah, that you can always compare figures and numbers that you get. The question would be, you would have to pretty well match the exposure period and the period off with the levels you find, and then I would think that probably many of the fibers you would see would be more recent acquisition to the lung, and as

A. (cont'd.) you...it's probably a fairly active, dynamic system. They don't just come there and sit, so you are continually replenishing, and once you've stopped the exposure, gradually the number are probably going to decrease.

So it might prove useful as a relative feature, but I certainly wouldn't want to say that because we find...for the sake of argument...a very low quantity of fiber in the family and you find some workers that have the same amount, or have something like that or even lower levels, that we ought to use the family levels as an indicator that well, this is the level we ought to try to maintain. I don't think it's really comparable.

The fiber size, I would suspect, in the home is going to be smaller. It's more likely that the fibers would have been broken up, so you may find that you need to do more electron microscopic work, that there won't be any optical fibers there because they are all small. I don't know. We certainly need to look at it though.

Q. You displayed at least one table this morning where you indicated that you had divided the family contact cohort up among smokers and nonsmokers, and compared their x-ray results?

A. Yes.

Q. I wondered whether you had given any consideration to also looking at the smoking habits of other members of the household?

A. We gave consideration to it, but we haven't looked at it, no. It's a possibility. It can be done. We could code them as to whether they are smoker versus nonsmoker household, or two out of five, or three out of five, because all of those that came in, anyway, we know the histories on.

Of course, those that have since died or left, it becomes less reliable information.

Q. One of the reasons I ask that is that some of

Q. (cont'd.) your former coworkers, Dr. Selikoff and Dr. Hammond, have recently published an article on what they call Passive Smoking and its Relationship to Lung Cancer.

Are you familiar with that article?

A. Roughly, yeah.

MR. HARDY: I have copies of it, Mr. Chairman, if you and other persons here would be interested in looking at it before I go on asking questions.

MR. LASKIN: Sure.

MR. HARDY: I'll just keep one.

MR. LASKIN: We never turn anything down, Mr. Hardy.

MR. HARDY: I don't have lots of copies, but...

DR. DUPRE: Do you wish to give that a number for reference purposes?

MR. LASKIN: I suppose exhibit twenty-five, Mr. Chairman. I haven't got Mr. Warren here to keep the numbers.

EXHIBIT # 25: The abovementioned document was then produced and marked.

DR. DUPRE: You are bringing his attention to it?

MR. HARDY: Yes.

MR. HARDY: Q. Have you seen this article before?

THE WITNESS: A. I have seen parts of it. Depending on what your questions are, I will refrain from..

Q. I'm not going to ask you any detailed questions.

A. Okay, if you are not going to ask me detailed questions, fire away.

Q. As I understand this article, it's a report... it's an article, I should say, by Drs. Hammond and Selikoff, about two studies they didn't do, but which are in the literature?

A. Right.

Q. One in Japan and one in Greece. This is their interpretation of those two studies. As I read it, to summarize very briefly, Dr. Hammond and Selikoff note that in both of these

Q. (cont'd.) studies increased risk of lung cancer was found for spouses of persons who smoke, even if the person themselves did not smoke?

A. Right.

Q. I guess my question to you would be, in light of evidence like this, could it be possible that smoking in households like those you studied might have some relationship to lung cancer risk, if any are found, in the family contact who are not smokers?

A. I would doubt that that would be the case. I think it could be looked at as a possibility, but I would want to look at whether or not...if there is an excess cancer, say lung cancer, mesothelioma is not associated with cigarettes, but if there were in the group as a whole excess lung cancers, before I would attribute it or say it even had a suggestion of attributing it to asbestos exposure, you would like to see...like with the x-rays...that there was a higher rate amongst the family members with longer exposure time.

In other words, you ought to see some kind of a dose response. It might be minimal, but you would like to see corroborating evidence more than just overall excess when you look at them lumped. So I would think that the cigarette smoking is more apt to be evenly distributed, and you can look at that. It would not parallel that sort of a finding, so I think it could be controlled for and I don't think the excesses...and I think they also go on here that they don't particularly agree with this observation of these two papers, and they point out some other problems with it.

I believe, and I'm not firmly convinced, or at least Dr. Hammond in here I don't think is firmly convinced, that this is necessarily an appropriate observation from sidestream smoking, that more information is needed on that.

So I don't think that we could attribute any excess cancer to sidestream smoking in this sort of an instance,

A. (cont'd.) especially when over half of our study group is going to be children who will have moved out of the index home and may be in with somebody else. Certainly we will try to look at the individual's smoking habits first, along with that.

In other words, if you are going to list priorities we have to be sure if we see an excess that it is not related to the individual's own smoking habits, and that will be a predominant factor over any sidestream smoking.

Q. You indicated that Drs. Hammond and Selikoff had some skepticism about the results of these other studies, and I think that's accurate. Perhaps we should look at page 452 of this article, and the next-to-last paragraph, just to see their own words on that subject.

> They state: "With so much at stake we hesitate to express an opinion at this time on whether, quote, 'passive smoking' increases the risk of lung cancer to a significant degree. It would take additional positive evidence, but perhaps not a great deal more evidence, to firmly convince us that this is so".

I just thought perhaps it's best to have their words.

A. Okay.

Q. In the same connection, would there have been smoking within the factory, the amosite factory, where these workers whose families you have studied were employed?

A. I don't think we have that information. I would suspect that there probably was smoking by some of the men, depending on the type of job they did. Certainly on break, I would suspect they were allowed to smoke. Depending on the type of equipment they worked, they may have required two hands and it's very hard to smoke at the same time...especially some of this type

A. (cont'd.) of equipment that they were using.

But I don't see that as...you know, we just don't have that kind of information. Many of the men did not work that closely next to each other, different shifts, and things like that, too.

Q. I gather though, that from a picture that Dr. Nicholson showed us last weekend, that insulators might commonly have smoked on the job?

A. I would suspect it's more likely with an insulator, if he is a cigarette smoker, to smoke on the job, than a factory worker. That's just a general opinion. Yes.

Q. Recalling another table you put up earlier today, you showed the breakdown in terms of lung cancer for smokers, nonsmokers, exsmokers, from the Selikoff insulation study.

As I recall the table, when we are talking about nonsmokers, the numbers are very small. I think it's point seven expected and four lung cancers observed.

A. Yes.

Q. Could it have been possible that some of those apparently-excess lung cancers may have something to do with passive smoking among insulators?

A. I suppose it's a possibility. I'm not sure that I would put it into a probability. You also have to think in terms of, you know, what other chemicals they may have been exposed to in those sorts of areas that may have contributed.

Of course, if it's significant, the effect from passive smoking as described in these two studies would have been far in excess of what was seen in the insulators. You know, the numbers you would expect would have been higher.

So in that sense what we are showing here would argue against some of the information here.

Again, the difference between a spouse smoking and working in an area which, for many of the insulators, for instance,

A. (cont'd.) is external, outside...there is much more airflow than, say, in a closed home...but it is a possibility.

I mean, you can't say that anything is not a possibility. If you have said that it's likely that this is the explanation, I would say no.

Q. You made a mention of other chemical exposure of insulators. Could you maybe just expand on what you were thinking of?

A. Well, we don't know what the effects of all the chemicals people work with are. So I would be concerned that if you are talking at very minute things, and possibilities as opposed to probabilities, the only thing that insulators handled...it's just like in the asbestos mines and mills. The maintenance people worked with gasoline and solvents and things like that, and if you say well, was there benzyne in that? You would say yes, and if you say well is it possible that benzyne could have been contributory in any case, is it likely? No.

So I think we have to...it's very difficult to take the person out of his whole milieu, and anytime we do see an association like this, you assume that what you are comparing to is going to be very similar.

I guess we would have to say that the control group that was used from the Cancer Society didn't control for sidestream or passive smoking, and since the asbestos group nonsmokers that were compared to show the excess, we have no reason to believe that their passive smoking would be any different than the control group, so both may have a passive smoking affect in it, but it should balance out by being comparable in the two.

I don't think that we will ever come to the point that we can explain every lung cancer that occurs.

Q. Although I gather that...

A. I mean, everybody can say that they have been

A. (cont'd.) exposed to cigarette smoke, passively. Therefore, you could argue that we have finally come to an explanation for every lung cancer we see, and I don't think that is a realistic approach to use.

Q. Although I gather these exposures that we have just been talking about to other chemicals and perhaps to passive smoking may be of greater significance when you are talking about the small numbers that we are talking about in comparing nonsmoking asbestos workers to nonsmokers in the general population. We don't have a big population.

A. Well, the general population, unfortunately we don't know what they are exposed to either. So generally the idea is, you make your comparison groups as alike as you can in everything but the variable of interest. Since you go out and if you look at Dr. Hammond's definition of how he selected out his cigarette smoking study, the control group, you know, it's a pretty good cross-section of blue collar workers, and sharing all of the exposures of blue collar workers. So you throw in a whole mishmash of stuff and hope that it's randomly distributed in both of your groups.

Until you see evidence that it's to the contrary, the only difference is one clearly has demonstrated asbestos exposure and the other does not. The one has an association with duration of exposure and onset, and the other just shows the usual age distribution.

So using humans as research subjects is a very difficult task because you cannot expose them to only one thing.

MR. HARDY: I don't believe I have any further questions, Mr. Chairman.

DR. DUPRE: Thank you, counsel.

M. Bazin, you are batting cleanup.

M. BAZIN: I'm not on strike.

CROSS-EXAMINATION BY M. BAZIN

Q. Dr. Anderson, at the beginning of your comments this morning you referred to...and I'm not sure if I'm quoting correctly...but you mentioned the problem that you detected was that the actual levels or the actual standards may not be maintained on the floor. I think you referred generally to those words.

Could you elaborate on what you meant by that?

A. Okay. The point I was trying to make at that time was that setting a standard is not sufficient to eliminate disease. That just because one has a number or a figure, that figure doesn't per se mean that people are exposed at or below that. So what I meant is, you can set any number you want, and what does or does not cause disease is what the man is being exposed to. That whether or not that's in compliance or out of compliance, if the man gets ill he has obviously had a significant exposure.

So my point was that there is a great deal of need to not only consider setting standards or having regulations or guidelines on whatever you want to use, but it's also important to make the measurements to see that they are in fact occurring at that level, and do it in a reliable manner.

That was the point I meant.

Q. Did you mean to say that as far as your experience was involved and what you may have found, particularly in the area where you investigated, that there was a problem between the standard and the compliance to the standard?

A. No, what I'm trying to say is, our studies have found an end point. We have found people who have been ill.

Now supposedly there were standards to prevent that. Somewhere along the line there was a breakdown. Now where that went, as a physician, if the man before me is ill with

A. (cont'd.) asbestosis or with mesothelioma, it doesn't really matter to my treatment of that man whether he has been supposedly exposed at two fibers per c.c. or at fifty. It makes a great deal of difference from the legal standpoint or the standard-setting standpoint. For that man, whatever his dose was, it was sufficient to cause his disease. The idea is, we want to decrease the disease.

Q. Therefore, you are insistent on safety at the workplace and the precautions that have to be taken to handle asbestos?

A. That's correct. Yes.

Q. I believe you said that asbestos can be handled?

A. Yes, I believe...

Q. If done safely?

A. I believe there are safe handling practices and safe uses of asbestos. Yes, I do.

Q. Coming back on your household contact study, just to clarify some of the numbers, the Chairman alluded to certain numbers and I would like to get these clear.

Originally, as I understand it, three hundred and twenty-four household contacts were made, and ...

A. That's how many were examined.

Q. That's how many were examined?

A. Yes, up to that point in time.

Q. It is stated in your article, which is tab two, that these household contacts were invited.

A. That's correct.

Q. Can you tell us how these people were invited?

A. Generally by a letter and by telephone contact.

Q. Who wrote those letters?

A. The Environmental Sciences Laboratory did the asbestos project.

Q. And followed up by the same people by way of telephone?

A. Yes.

Q. Then in 1979, there are about...and it's tab seven...there are seven hundred and seventy-six people...just above seven hundred and seventy...that again were examined?

A. Right.

Q. Are you able to tell us if the original three fifty are in those seven fifty, or how many are?

A. Oh, yes. All of them are.

Q. Okay.

A. What this is, it was a standing clinic where people would come in. We had roughly eight or nine weekend clinics a year, and the people in the three twenty-six are also the people in the seven hundred. All it is, is there is an additional four hundred new people who came into the clinic and we just kept summing up the total.

Q. That is what has been examined out of three thousand, one hundred household contacts that were identified. Is that correct?

A. I'm not sure that's right. I may have...it's something like that. I don't know exactly what the total is, but there is roughly between two and three thousand total in that cohort. I wouldn't say it's three thousand, one hundred, but it's somewhere around there, yes.

Q. If we are talking about household contacts, of sixteen hundred workers...

A. Right.

Q. ...doesn't that number appear low?

If the family by then, in the U.S.A., was composed of at least four people, or an average of four people...

A. Okay. What you have to understand is, these are not average people. What they are, as I said earlier, many

A. (cont'd.) of them were single men who came and worked there but did not live with their family.

In other words, very many of them...I would have to look it up...either were single or were married and did not have their family, or they were married and of an age where they were just with the wife and the husband and there were no young children at home. So it's a gamut, yes.

Q. On the other hand, you have included some cousins, which would infer a large type of family, and other siblings, up to about twenty percent. So it would mean that there would be more than just the immediate family involved, is that correct?

A. A sibling could be that a man came up and lived with his brother. Something like this, where...into that area.

We are fairly confident that we have identified all the people that lived with them at that time. You have to remember that in many cases we are talking a very short period of time, a working life period when a person became eligible for the cohort, of over half of them less than a year. So unless we caught that cut in time properly, the average household at that time would have to be taken into account, and the age of the man in his various circumstances...he may have subsequently gone on and had more children, or the children may have moved out of the house and he had, on the average, a family of four. But that doesn't mean the family of four is living together at that time.

Q. If we look at tab two, page 313, and I don't want to belabour this point, but I just want to get that three hundred figure straight. That's table two. You have the relationship to your index worker, I guess is what you call him.

You have the children, you have the wives and daughters, then the children, then brothers and mothers, and others,

Q. (cont'd.) including cousins.

A. Right.

Q. Again, doesn't it appear to you, given the restrictions that you mentioned, that the three thousand or between two and three thousand replies would not constitute the whole potential cohort?

A. No. I don't think you can imply that this is an unreasonable number of people to have as part of the cohort, or that there would have been more or less than that. I mean, I feel confident that having talked to the person and having gone through the city directories which list how many people are living in that household at that time, that we have identified upwards of ninety-some percent of who was living in that household at the time of the exam.

Now, under the cousins, etc., others, of which there are thirteen, that would be unusual circumstances where somebody came up and probably lived with somebody else, or a cousin happened to be living in the household at the time.

Q. Given those comments, your testimony is to the effect that the only measurements made into these households were some visits by Dr. Nicholson? Is that correct?

A. That's correct.

Q. There was no other effort to measure what actual exposure may have been in these households?

A. That's correct. You have to remember that the plant closed in 1954, and the study was done twenty-five years later. Many of the houses they lived in are no longer in existence.

Q. And the records for the exposure in the plant were also not available at the time of your inquiry, is that correct?

A. Exposure records are just not available in this plant at all.

Q. As a physician, Dr. Anderson, one final

Q. (cont'd.) question, is it your opinion that in the mining and milling end of the asbestos industry that smoking should be prohibited in the workplace?

A. As a physician treating patients, I believe every patient I see, especially those exposed to asbestos, should not smoke cigarettes.

M. BAZIN: Thank you.

DR. DUPRE: Thank you, M. Bazin.

Commissioner's hour. Dr. Uffen?

DR. UFFEN: Twice today you have used the expression 'safe handling procedures'. The word safe can have different meanings.

Would you like to explain what you mean, regard as safe?

THE WITNESS: I guess what I would call safe would be handling procedures or activities which would, under the current handling procedures, be considered appropriate and would keep exposures to a minimum.

DR. UFFEN: Minimum attainable or minimum detectible?

THE WITNESS: I would say minimum attainable.

DR. UFFEN: I see.

THE WITNESS: If you are handling it appropriately, that's going to be your best available mechanism at that time.

DR. DUPRE: I'll just ask a followup question, linked to the same statement that you made in your dialogue with M. Bazin.

Could you elaborate a little bit on what you had in mind when you referred to safe uses of asbestos? You've talked about the importance of safe handling procedures and safe uses of asbestos.

THE WITNESS: Well, I don't think I can give you a list of what would be considered safe uses. I would feel that uses where you can apply or have some control over where the

THE WITNESS: (cont'd.) asbestos goes or how it is handled...for instance, I think you can train workers appropriately and give them proper equipment and knowledge so that they can handle the material...to go back to that...as safely as possible. That if anybody is going to handle it safely, it would be these individuals. If you are going to use it at all, properly trained, equipped, in a proper facility, with proper ventilation, it could be used.

I guess I would be concerned about asbestos in consumer products where the only training is perhaps a warning on a label, or a situation like with the hair dryers, something like this. I think that carries with it an inherent risk to the user who is not properly informed or properly trained. So I guess if we did it in a generic sense, I would say under situations which can be controlled, where access to the material, at least the raw material, can be controlled to the extent that you can tell the individual and train the individual. That would be what I would consider to be a potentially safe use.

I guess I would also have to say an appropriate use. In other words, if there is something that's better than that material, and asbestos is a known hazard and there is a replacement material for it that does the job, then I would think that you ought to opt for the material that does not have the hazard.

But I believe there are...I'm not an asbestos-use expert, but I would suspect there are uses that it is going to be extremely difficult to replace asbestos in, and in those circumstances, as long as you can have adequate monitoring and control, I think it can be handled safely. It always has the potential for being a hazard and a problem, and I think in the past...going back to my second slide there from Montague Murray... they knew how to handle it, they knew how to suppress dust, but somehow or other that didn't seem to get put into practice.

A. (cont'd.) I think that's where we have to be very careful.

I would say adequate monitoring, surveillance, education of the workers - if all that is followed out, and you can be certain that it is being done right, then I think you are going to have your safe condition.

Now that may be an ideal situation that never occurs out in the environment, but that's what I mean by safe use of it.

DR. DUPRE: Now who is to suggest at least the theory that there is a safe threshold with respect to exposure to asbestos?

THE WITNESS: No, I'm not saying there is a safe threshold. But I think if you are...there may well be a relative risk, if we talk in those terms, that will get down so low that any excess one would see would be moderate, that you'll have a very small work force who will be adequately protected, that if you are working in a space suit, you are going to have a difficult time with a controlled air supply, of getting those fibers into your lungs.

I'm not sure that under all circumstances the best available precautions are always taken, and that there is always human error, and that becomes a problem.

Now that's a decision that's a nonmedical decision, nontechnical. But technically, I think you can handle the material and I think there are...this would be for the engineers to tell you...situations where it is wholly appropriate.

We do have other carcinogens, at least in the U.S., that are under use, that are very closely regulated and licensed to handle them. We have certainly used radiation and that doesn't mean that because x-rays from diagnostic radiology can be associated with increasing dosages and therefore in various studies found to be adding to our body burden, that we should do away

THE WITNESS: (cont'd.) with those things and that the gain from that overweighs the risk.

So I think there will be similar situations with asbestos, but I do think it needs to be tightly looked at and controlled.

DR. DUPRE: Dr. Mustard?

DR. MUSTARD: I'm having trouble trying to weigh the comments we have heard from you and from Dr. Nicholson about the question of the amount of exposure people have received in their homes. I'm trying to recall, and I'm not sure whether Nicholson told this or not, if he went and sampled other houses in that area and looked for asbestos fiber in the dust. What levels were found in comparison to the homes that were looked at, do you recall?

THE WITNESS: I believe it was looked at in a number of homes, but it was more of a qualitative than a quantitative sort of thing. It was in a bulk sample, it was not an air sampling type of mechanism. I don't believe there were a large number of homes done, and of course it was many years afterwards, so that becomes...at the point, at least from my standpoint, what I wanted to know is, is there still settled dust, amosite, in these homes and this is just about the only source. Amosite is not commonly found, although chrysotile is more commonly found in settled dust.

DR. MUSTARD: Supposing I asked you to sample one hundred homes in that area for which there is no record of any workers from as asbestos plant having lived in the home. Would you have found any amosite in the settled dust in those homes? Has that been done, do you know?

THE WITNESS: Not a hundred homes. I believe there were a few. You would have to ask Bill. I wouldn't want to...at least my recollection is that it was not found in some of the other homes. Now I may be wrong. I didn't do the work.

THE WITNESS: (cont'd.) Much of that was done as part of a worker mortality study, not as part of my family clinical study.

DR. MUSTARD: My second question is, could asbestos-like material have entered the homes through other routes? I can take you to smaller towns around here and there is a hardware store, and you can buy bags of asbestos just off the shelf and take them home.

It seems to me that's a very good supply of asbestos to take into the house, which would be probably much greater than what I would take home in my workclothes.

What was going on back in those days, in the area which you were doing your studies? Was asbestos available in hardware stores in those days, in bags?

THE WITNESS: I really don't know.

DR. MUSTARD: Was that looked into?

THE WITNESS: There was other potential sources, but we didn't specifically ask 'did you work with asbestos in your home'. I believe back in those years it was before drywall installation and most of the home asbestos products, I believe, that I've seen, are spackling-type surface treatments. I don't know, maybe some of the industry people could tell us, how far back those types of commercial products go. But I don't think they go back that far.

DR. MUSTARD: Let me pose another question. In the testimony that we've heard it has been indicated that mesothelioma, as much as maybe one-third, cannot be identified as being caused by asbestos exposure. Is that correct?

THE WITNESS: In some studies, yes.

DR. MUSTARD: This creates a problem in terms of what is the cause of these other mesotheliomas, and let me pose it in this way: If we are engaged in a major control system in our schools and we are told we can't really do studies about the

DR. MUSTARD: (cont'd.) exposure of people in the schools historically in the past in terms of developing mesothelioma, yet if you think about it I guess the use of asbestos in buildings certainly was going on in the fifties, and various things, so that it is possible that some of those people, I suppose, exposed at that time could be coming down with mesotheliomas.

Now, when you were asked this question you said it couldn't be done, but surely there could be some way you could track into that to find out whether some of the mesotheliomas that are occurring in people today, which you cannot show an exposure for, can be traced back to possibly school exposures. Has anybody looked into that?

THE WITNESS: I'm not aware that anybody has. I don't want you to get me wrong, to say that I don't think...that I think it's impossible to do. I think it's a very difficult, time consuming, costly thing to do, and I think there are very many epidemiologic problems in it as to ruling out is it...how do we isolate schools. We are going to have to identify a population of people who, one group went to school known to have asbestos-containing materials, which was in probably poor repair, and who subsequently left, stayed in the same area and did not have any potential for occupational exposure.

Then you have to have another group of children or individuals in a school that did not, that you know as a fact did not contain, thirty years ago, any asbestos material, and that they didn't ever work with any of these things.

It's theoretically possible. I think, you know, it would be interesting to see somebody try, but I think that is extremely difficult. I think whether or not mesotheliomas are associated with asbestos, a lot depends on what kind of history you have available to you and who that history comes from and what you accept as being asbestos exposure.

Now, if you make a very liberal definition, then

THE WITNESS: (cont'd.) many more of your cases in historical studies may have asbestos contact. But in general what the various series have done is to try to look for fairly substantial exposures and not just circumstantial.

As I say, everybody has gone to school and if they haven't gone to school, they have visited a school. If you would take a child, you would have to follow them and say well, do we have a group of kids who went into this gymnasium, or this group who did not, what about when they went to visit a school that had asbestos in it, what about other public buildings they may have worked in that had spray asbestos in them, is this any more or less of an exposure?

It becomes very difficult. I think carefully thought out you might be able to do it, but even at that point it would still be difficult to design it such that it would be impeccable.

I guess that...

DR. MUSTARD: That's true for the other studies as well...

THE WITNESS: Oh, absolutely. But the whole point is you have very, very many other studies, all of which, although the numbers may vary a little bit, come to a general conclusion. So one school study would beget, hopefully, or you would need to have others and especially that would be a difficult thing.

DR. MUSTARD: Surely it's one case where you don't just have the shipyard workers, you have full cohorts theoretically exposed during that time frame...large cohorts, with records throughout the system. I guess I could argue that, that the cohort to look at could be quite substantial...

THE WITNESS: Well, it's...yes. That's possible, but again, the more substantial the cohort, the more difficult it is in tracing these people over thirty years.

THE WITNESS: (cont'd.) Now it may be more readily attainable here in Canada than it is in the U.S., but...

DR. MUSTARD: Now, we have too many people go across the border into the United States.

THE WITNESS: Well, I can tell you the problems that we had tracing a relatively small group of workers' families, and you can realize that once you find one, most people keep in touch with their kin. But when you have John Jones who lived on 1 East 32nd Street for three years, and then the family moved on, and you are going to have names changed...you can't just study male children, so you are going to have females who by the age of thirty are now married, and how are you going to find where they went unless when they were in school...I don't know, here I guess you do have their health number, or something like this, you might be able to trace.

But if all you have is school records that list the name and address of a kid in third grade, and you then have to match that to see how long they stayed at that school, and then try to find a whole group, and you can't be satisfied with less than ninety-some percent, find them thirty years later, what has happened to them. - let me tell you, that is one big job.

DR. MUSTARD: Then there are chances of...you said in your comments that in Wisconsin you are using EPA-approved substitutes?

THE WITNESS: No.

DR. MUSTARD: I had that impression, in terms of replacement in fire protection.

THE WITNESS: No, it's not...what we have is a list of EPA encapsulation materials. They have a testing laboratory which has been testing all of the products that come on the market to see are they...

DR. MUSTARD: This is for encapsulation?

THE WITNESS: This is for encapsulation.

DR. MUSTARD: I see. They are health rated as well in terms of their potential health effects, do you know?

THE WITNESS: I don't think that that necessarily is true. Supposedly yes, but they are primarily for will they do the job they are supposed to do.

DR. MUSTARD: As an environmental health officer, what assurance can you give the school boards in Wisconsin that what they are using for encapsulation will not be a potential health hazard some fifteen years down the road?

THE WITNESS: I think I can give a pretty good assurance.

DR. MUSTARD: Based on what evidence?

THE WITNESS: Based on looking at the chemical composition of these compounds that are primarily hard, solid polymers, that unless they were to burn, if you had a fire in the school they might present a potential problem just as the plastic furniture and rugs, etc., would. But I don't think that they, as a completed product, represent a problem as far as inhalation.

DR. MUSTARD: You would have predicted urea formaldehyde would have created problems, when it was introduced, because of its chemical structure?

THE WITNESS: I don't think...you mean as far as its carcinogenicity?

DR. MUSTARD: I am trying to get at the question of whether you can predict from chemical structure about the potential health hazard, easily?

THE WITNESS: I think from chemical structure, as well as the nature, as to is this a volatile, what is the source of exposure. I think you can, to a certain degree, you can't be one hundred percent certain. Similarly I would say that most of these compounds you can feel fairly certain that in their completed form they are going to be reasonably safe.

THE WITNESS: (cont'd.) Now, will they ever? There is no such thing as never present a risk, but that is one consideration. But I guess what has to be weighed is that versus the asbestos falling down, and it's a material that we know represents a problem.

DR. MUSTARD: My final question, and perhaps some of my colleagues will understand the reason for it now, you are a recent graduate of a medical school?

THE WITNESS: Yes.

DR. MUSTARD: In a period of time when understanding of envirnomental health hazards has certainly advanced, and I gather from your comments you are a little concerned that you didn't get as much exposure to it as might have been desirable.

As perhaps a question, two questions, one: Has this now been improved in the institution in the United States in terms of the educational physicians? Perhaps you would like to answer that question first.

THE WITNESS: Improved...

DR. MUSTARD: Enhanced.

THE WITNESS: Enhanced?

DR. MUSTARD: The education of medical students?

THE WITNESS: I think it probably has, compared to what it was. If it has the proper significance or the proper enhancement, I would have to say no. Of course, you have to recognize I'm very much of an advocate for occupational and environmental health, and have been pushing for some time to have this increased in the curriculum.

But certainly if I use Mount Sinai and the University of Wisconsin as examples, the hours devoted to this sort of information is certainly...well, there are actual hours now where when I went to school it was included under respiratory disease for the dusts, for other parts of toxicology. Whereas now there is a specific series of lectures on occupational

THE WITNESS: (cont'd.) environmental issues.

DR. MUSTARD: Can you do anything more to enhance their education in this field? Enhance the education of the student, or do you think it's satisfactory at Mount Sinai?

THE WITNESS: Oh, I don't think it's satisfactory. Again, from my perspective I think they should know more, and I don't think they have a sufficient appreciation. I think it's very difficult. You have to weigh what is needed in a medical education and where this fits in in relation to the other things. That's where these issues get sorted out, and certainly I think you need more in education. I wouldn't just single out the medical schools. I think there is also a need for more of this sort of information in the public school, grade school on up, just as we get into...you have health education courses in classes. I think you ought to include in that environmental occupational factors.

I would like to see it increased all the way up. Certainly there are not enough people going, or professionals being trained either in occupational environmental medicine as specialists, or enough technicians to go out and do monitoring in the field.

Industry has a hard time getting enough industrial hygienists. So all these fields...and those people also need to be educated into health hazards.

Then of course we have worker education, too.

DR. DUPRE: Counsel, any more questions?

MR. LASKIN: No, Mr. Chairman.

DR. DUPRE: Well, Dr. Anderson, on behalf of all of us here may I think you very, very much for your visit here with us today, and for your very useful comments.

THE WITNESS: Thank you.

DR. DUPRE: Counsel, we reconvene tomorrow morning at ten a.m.

MR. LASKIN: Ten a.m.

DR. DUPRE: Is that correct?

MR. LASKIN: That's correct.

DR. DUPRE: Ten a.m. it is, then.

THE INQUIRY ADJOURNED

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THE FOREGOING WAS PREPARED
FROM THE TAPED RECORDINGS
OF THE INQUIRY PROCEEDINGS

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